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## STATE MEDICAL JOURNAL

*Medical and Chirurgical Faculty of the State of Maryland*

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# Maryland STATE MEDICAL JOURNAL

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VOLUME 2

October, 1953

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## **Blue Cross-Blue Shield Enrollment Period for Medical and Chirurgical Faculty Members and Employees During November, 1953**

The annual Blue Cross-Blue Shield enrollment period for Medical and Chirurgical Faculty members and their employees will take place during the month of November. During this time, applications will be accepted for new members and for present subscribers desiring a change in type of coverage. The latter part of October you will receive a letter from Blue Cross-Blue Shield presenting information covering the details of enrolling along with explanatory material and application cards.

Blue Cross is designed to pay the hospital bill when a subscriber is hospitalized. Blue Shield is the partner plan of Blue Cross. It provides medical and surgical benefits in the hospital when such care is needed.

Under the Blue Shield Plan, single subscribers whose total income is \$3,000 or less, or married subscribers whose total income is \$4,000 or less are eligible for full service benefits. If your income exceeds these amounts, your doctor will ask you to pay the difference, if any, between the Blue Shield allowance and his usual fee.

For additional information concerning the advantages of the Blue Shield Plan, please consult the folder which will be sent you by the Blue Cross-Blue Shield Office.

While the majority of Faculty members are probably familiar with the details of Blue Cross, a summary of the main features of the Plan is presented for your convenience.

For subscribers using semi-private rooms, Blue Cross pays the bill in full for the customary hospital services regardless of cost. These services are:

- a. Room, meals, special diet.
- b. General nursing care.
- c. Use of the operating room.
- d. Anesthesia given by hospital employee.
- e. Standard drugs and medication.
- f. Laboratory examination (when necessary part of in-patient care).
- g. X-ray examinations.
- h. Electrocardiograms.
- i. Physiotherapy.
- j. Casts and dressings.
- k. All other customary hospital services.

Subscribers using private rooms receive a credit of \$7.00 a day toward the room charge plus a credit of 75% of the charges for all customary hospital services.

As in the case of Blue Shield all basic information concerning Blue Cross will be contained in folders which will reach you direct from the Blue Cross Office.

Blue Cross-Blue Shield is the nation's largest non-profit health program with over 44,000,000 members in 87 Blue Cross Plans protected through the cooperation of over 4,500 hospitals and with over 23,000,000 Blue Shield subscribers protected through the cooperation of the nation's medical profession.

Continued support of the Blue Cross-Blue Shield Plans is still the best means of enabling citizens of Maryland to pay for their health care on a voluntary basis. The success of Blue Cross-Blue Shield is universally used as a most effective counter-proposal in discussion and debates wherever the need for some enforced health program is advocated. Blue Cross-Blue Shield, operating as they do on a non-profit basis with the cooperation of the hospitals and medical profession has proven to be the most inexpensive method of providing comprehensive benefits to persons who wish to maintain freedom of choice and opportunity in managing all needs pertaining to their health.

\* \* \* \* \*

#### *DOCTORS IN KOREA TO AID MEDICAL REHABILITATION*

The AMA Washington Letter, No. 34

U. S. Armed Forces physicians in Korea will be utilized to help in the rehabilitation of South Korea's medical services, according to a Defense Department directive. The department explained that uncertainties in Korea require retention of American troops, with the usual ratio of doctors. When these physicians can spare the time from their military duties, they will be expected to help in the re-establishment and expansion of medical teaching facilities, hospitals, public health, and "other programs required for the betterment of the health and welfare" of the population. Defense Secretary Wilson, however, emphasizes that the new policy may not be used "to justify the retention of medical personnel in the Far East beyond their established tour of duty nor as authority to staff the required military facilities in that area beyond that necessary to carry out the military medical mission. . . ."

\* \* \* \* \*

#### *FEDERAL SCHOLARSHIPS*

The AMA Washington Letter, No. 34

Defense Department officials are at work again on a proposal to have Congress authorize federal scholarships to *students in medicine*, dentistry, nursing, and several other health fields. Recipients would be required to spend a specific amount of time in military service following completion of their education. The idea has been suggested previously in a number of bills offered in Congress, but so far nothing has come of it.



## PRESENTATION OF VOLUME<sup>1</sup> CONTAINING THE WRITINGS OF DR. HARVEY G. BECK<sup>2</sup>

WALTER D. WISE, M.D.<sup>3</sup>

Mr. Chairman, Mrs. Beck, Mrs. Mahan,<sup>4</sup> Ladies and Gentlemen:

In a few minutes you will hear the first Harvey Grant Beck Lecture by Dr. T. S. Danowski, a Lecture generously endowed by Mrs. Beck. (See pages 530-537.)

Dr. Beck was born in York County, Pennsylvania on August 15th, 1870 and his life came to an end in Baltimore on October 30th, 1951. He was active to within a few days of the last. He lived a long and most useful life and I believe he enjoyed it fully. He enjoyed it because, in addition to the affection bestowed upon his fine family, to whom he was devoted, he loved people and he loved work. That he was an indefatigable worker is proven by his whole career, which encompassed the threat of being a farmer but instead he chose to teach school and later took up the study of pharmacy and still later he studied medicine, being graduated by the College of Physicians and Surgeons in 1896. He was made a Demonstrator of Clinical Laboratory Diagnosis in 1898 and by 1916 was Professor of Clinical Medicine and remained in this position at the University of Maryland for 30 years, being made Professor Emeritus in 1946. During most of his career he was on the staffs of Mercy, Sinai, University, Church Home and Union Memorial Hospitals and, when the South Baltimore General Hospital was made a general hospital in 1918, he was named Chief of the Medical Staff and retained this position for many years, resigning because of pressure of other work.

The list of medical societies and other organizations, to which he belonged, covers two typewritten pages. I wish there were more time to detail his career. He was active in visiting clinics in this country and abroad, doing post graduate work in his early years in Vienna and Berlin and later visiting various hospitals in England, France and other countries.

We are in a room dedicated to a famous physician, who said, "the master word is work" and certainly Dr. Beck agreed with this philosophy. He soon established a large practice which increased as the years went on, eventuating in his private clinic, which was the only one of its kind in these parts. His being dedicated so wholeheartedly to his work did not preclude avocations such as becoming an enthusiastic and expert photographer and upon occasion lapsing into poetry. His medical writings were started early. The first record we have being of one published in 1899. Articles then appeared in journals at rather frequent intervals and in the meantime Dr. Julius Friedenwald, Dr. Beck and Dr. Hubert C. Knapp published a book in 1904 on "Clinical Laboratory Diagnosis."

The early papers had largely to do with laboratory techniques but as early as his twelfth paper published in 1909, he showed interest in the pituitary gland and his interest in endocrinology remained with him all his life. He published numerous papers dealing with this subject. Perhaps his next greatest interest was in chronic carbon monoxide poisoning, his first publication on this subject being in 1924 to be followed by many others. His writings, mostly on scientific subjects, total 70 publications, the last one being published in 1950, this paper being in the nature of a reversion to his first interests and was on "Orthostatic Albuminuria."

Dr. Beck was a man of determination, ability, almost limitless energy, thoroughness, gentleness and great kindness. He was fond of this Faculty. It is nice to have his writings in one volume prefaced by a short biographical account of his life prepared by loving hands. It is a great privilege to present to the Faculty this volume of the seventy publications, which are an index to Dr. Beck's interests and are the proof of some of the characteristics I have mentioned. Others, such as his modesty and cheerful laugh, could be appreciated only by knowing him.

<sup>1</sup> Gift of Mrs. Harvey G. Beck.

<sup>2</sup> Presented on Tuesday Afternoon, April 28, 1953, Osler Hall, Baltimore, Maryland, Annual Meeting, Medical and Chirurgical Faculty. (Part of 1953 Transactions.)

<sup>3</sup> Past President and Past Secretary, Member of Council, Medical and Chirurgical Faculty.

<sup>4</sup> Mrs. Archie I. Mahan, daughter of Dr. Beck.

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# Scientific Sessions

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## INTERRELATIONS OF THYROID AND IODINE METABOLISM<sup>1</sup>

T. S. DANOWSKI, M.D.<sup>2</sup>

The need for precise indices in studies of thyroid physiology is emphasized by the known limitations of basal metabolic rate measurements, lipid analyses, magnesium determinations, radioactive iodine pickup, incorporation, or excretion studies and even responses to therapy in evaluating specific patient situations. Enough data have now been accumulated on the utility and limitation of protein-bound or serum precipitable iodine levels in establishing the status of thyroid function to permit certain deductions concerning the interrelations of this gland and various aspects of health and disease.

### I. SERUM IODINE FRACTIONS IN HEALTHY ADULTS

It is known of course that the thyroid gland possesses an unique, or at least a highly developed, capacity to remove inorganic iodine from the blood circulating through it. This halide is then coupled with the amino acid tyrosine; molecules of halogenated amino acids are united to form the thyroxine molecule. This substance, in an as yet unidentified linkage with protein, is either the hormone of the thyroid gland or, in view of the metabolic effects of triiodothyronine, a closely related precursor. Thyroxine enters the colloid for variable periods of storage prior to

being released to the circulation, and to the tissues. In the plasma it is under ordinary circumstances present in the protein-bound or serum precipitable fraction of iodine. One must keep in mind however that the plasma may also contain other iodine moieties such as a non-thyroxine protein-bound iodide and others.

With an intake of inorganic iodide limited to that present in an ordinary diet the inorganic plasma or serum iodine fraction is about 1 gamma per cent. Under similar circumstances the protein-bound or serum precipitable iodine (PBI or SPI) in euthyroid healthy individuals makes up some 4 to 8 gamma per cent. It has been established that during intervals of at least one and one-half to two months the protein-bound iodine remains relatively constant in any one individual (1). In one of the subjects in that particular series for example the variation from week to week was one-tenth of a gamma per cent or less. It would also appear that in adult females a tendency toward slight fluctuations may be evident during the ovulatory-menstrual cycle. These alterations do not vitiate the relative constancy of the serum precipitable iodine concentration, since the change is only a fraction of a gamma per cent. The trend toward an intermenstrual rise or a menstrual fall does nonetheless suggest an interrelationship, though it need not be directly endocrine in character.

Much more profound changes in serum iodine fractions, on the other hand, may be encountered, during pregnancy. Heinemann and co-workers were the first to report that during gestation unequivocal rises in the protein-bound

<sup>1</sup> Harvey Grant Beck Memorial Lecture Annual Meeting, Medical and Chirurgical Faculty of the State of Maryland, Tuesday Afternoon, April 28, 1953, Osler Hall, Baltimore, Maryland.

This paper is a part of the 1953 Transactions.

<sup>2</sup> From the Department of Research Medicine and the Children's Hospital of Pittsburgh, University of Pittsburgh School of Medicine. Supported by grants from United States Public Health Service.

iodine to concentrations distinctly above the usual range in healthy non-pregnant adult females can and usually do occur (2). We have shown that the thyroxin is also elevated (3). It is clear, however, that values of, for example, 7 gamma per cent for protein-bound iodine during pregnancy could represent a distinct elevation above prepregnancy levels of 4 or 5 gamma per cent. The mechanisms or adjustments whereby these rises are produced remain essentially obscure. The theoretical considerations are of course clear. The possibility that these rises represent the results of hemoconcentration can be readily dismissed in view of the expanded blood volume characteristic of pregnancy. Hence, the increased circulating protein-bound iodine and thyroxin levels must represent either a) an increase in the rate of production and release of thyroid hormone, b) a rise in the organic iodine binding capacity of serum proteins, or c) a decrease in the rate of its peripheral utilization or disposal. The pregnant patient however disposes of exogenous thyroxin at rates undistinguishable from those found in non-pregnant young adults (4). This excludes the last of the three possibilities but does not define the role of the remaining two in producing the rise in PBI during pregnancy.

Spontaneous abortion on the other hand is frequently characterized by an absence of such increases in protein-bound iodine and thyroxin (5). Suggestive evidence is available indicating that in some of these patients desiccated thyroid or parenteral thyroxin may forestall this event (6). These correlations provide support for the time-honored obstetrical custom of thyroid administration to patients with infertility or abortion. They cannot be used however to explain all instances of prematurely terminated pregnancies since abortions do occur despite the presence of a characteristic pregnancy rise in PBI and thyroxin.

The course of the PBI following delivery is worthy of some comment. In a series of 95 women the PBI had returned by the fifth to seventh

week to values indistinguishable from those characteristic of non-pregnant females of the child-bearing age (7). Far more interesting however is the finding that in a large number of such subjects at a later date abnormally low PBI levels were present during the first year following delivery. These cannot be attributed to depressed albumin or globulin concentrations and hence might well indicate decreased thyroid activity and explain at least some of the postpartum fatigability ordinarily attributed to increased household burdens. There is no evidence one way or another that breast feeding, amenorrhea, or other events etiologically or temporarily related cause or prevent this drop, though this may well be so. Further observations are necessary however to determine whether or not these changes may represent temporary depression of thyroid hormone production or release, or a continued high rate of PBI degradation or excretion, or both.

## II. OBSERVATIONS DURING INFANCY AND CHILDHOOD

Random sampling of the newborn population of a large obstetrical hospital has indicated that at term or shortly thereafter the serum protein-bound iodine levels are as high or higher than those present at that time in the mothers, individually or collectively (8, 9). During the first week of life the infants as a group manifest a further rise which is statistically significant. This may be solely related however to the loss of body weight and decrease in fluids which characterize this period. Subsequently the protein-bound iodine drops to ranges which fall within the adult euthyroid values, but at a level which exceeds the mean characteristic of the latter group of subjects. It seems reasonable to suggest that growth and development in early infancy is either facilitated or mediated by increased levels of circulating protein-bound iodine and thyroxin.

In view of the above speculation the obvious possibility suggests itself that premature infants might benefit by thyroid administration. Though this has been tried in the past, only small doses

of thyroid were given (10). We have recently completed a study based on the premise that the requirements of the newborn infant for thyroid on a per kilogram of body weight basis may be much greater than those of the adult (11). The data suggest that this is indeed a fact and that the premature infant can be given up to 3 grains of desiccated thyroid or 0.2 mgm of thyroxin per day before manifestations of thyrotoxicosis appear. There is no clinical evidence that thyroid supplements in this larger range of dosage benefit the premature infant. These studies do indicate that adequate replacement therapy in cretinism requires thyroid in dosages larger than a fraction of a grain.

In another series of studies in diabetic and non-diabetic children certain provocative correlations have been demonstrated. Amongst these is the finding that the lowest protein-bound iodines in boys were present in the 12.1-14.9 year age group whereas in girls this was encountered in those who were between the 11.0-12.1th years of life (12). This points to the possibility that adolescence is associated with lowered levels of circulating protein-bound iodine. Another finding in this same series worthy of mention is related to intelligence quotients. In a small series of children intelligence quotients below 90 were associated with a significantly lower protein-bound iodine than that present on the average in subjects with intelligence quotients above 110. Perhaps the most reasonable interpretation which can be advanced is that there may be a correlation between this fraction of iodine and a generally inferior protoplasm. There is no reason to consider seriously the possibility that thyroid therapy would favorably affect the intelligence quotients of these patients, though this is being tested.

### III. PROTEIN-BOUND AND THYROXIN IODINE IN DISEASE STATES

#### *A. In Hyper- and Hypo-Thyroidism*

The most distinctive and, clinically, most useful correlations between protein-bound iodine

and thyroid function are encountered in myxedema or hypothyroidism and in thyrotoxicosis (13, 14, 15). Assuming the absence of factors which will be identified later, a close correlation is present between the degree of thyroid function, the level of metabolic activity, and the concentration of circulating protein-bound or serum precipitable iodine. The euthyroid range of this serum fraction lies within 4 and about 7.5 or 8 gamma per cent. Values below this range usually indicate thyroid underfunction; those above, point to thyroid over-activity. It should be emphasized however that in evaluating any particular analytical result the error of the method must be kept in mind. Since this is about 0.3 gamma per cent, and at times probably greater, it is obvious that figures beyond the decimal point at the limits tentatively set for either end of the scale will at times be of questionable validity. However, if these equivocal situations are excluded, the protein-bound or serum precipitable iodine is usually a highly reliable index of thyroid activity. This has been established by extensive and well-documented series of clinical situations in which the symptoms, physical findings, and laboratory analyses have been correlated with changes following upon the use of iodine solutions, goitrogens, or radioactive iodine with or without surgical therapy (13, 14, 15). It should be pointed out however that the circulating protein-bound iodine would not rise if the peripheral utilization increases at the same rate. Fortunately, from the view of the clinical problems encountered, a lag appears to be present. Hence in most instances the protein-bound iodine remains elevated above euthyroid limits despite the increased peripheral disappearance of the hormone. With diminution or cessation of hormone production on the other hand the circulating levels invariably decline and even reach zero. However, in evaluating any particular serum precipitable iodine concentration it should be remembered that a delay may well be present in the appearance of changes in the serum. This is certainly true in the case of complete thyroidec-



tomy or in the withdrawal of exogenous thyroid from subjects with total myxedema. It may take as long as six weeks before the last detectable vestige of the serum protein-bound iodine disappears.

#### *B. In Thyroiditis*

Only a limited number of observations have been made of the protein-bound iodine levels in patients with acute thyroiditis. In two out of three such individuals observed in this laboratory elevations into the so-called hyperthyroid range have been encountered (16). In one instance a value of 12.3 gamma per cent was found. Similar changes have been recorded by others (17). In none of these was there any clinical evidence of hypermetabolism. It seems probable that during the inflammatory process thyroxin and organic iodine compounds other than thyroxin are released into the circulation and augment the protein-bound iodine. It is not known what proportion of the rise is to be attributed to thyroxin and what to non-thyroxin compounds. The ultimate risk in these subjects is hypothyroidism or myxedema, as a consequence of the scarring and fibrosis which occur, with the serum protein-bound iodine low or absent.

#### *C. In Hypoproteinemia*

Since the circulating organic iodine fractions consist of combinations of iodine compounds with serum proteins, it is not unexpected that changes in the latter may influence the concentrations of protein-bound iodine. Peters and Mann have demonstrated that this is indeed the case in some but not all instances of hypoalbuminemia (18). Certain patients with nephrosis, liver disease, and other disorders with associated changes in the serum proteins have been found to have abnormally low levels. This does not appear to be, however, a sign of hypothyroidism, but rather a reflection of changes in the plasma transport system upon which the level of the iodine fractions depends.

#### *D. In Miscellaneous Clinical States*

It has been previously reported that in leukemia the protein-bound iodine is normal or elevated (19). We have found that the PBI and thyroxin level is within the euthyroid range (20).

In a series of 50 cardiac patients in congestive failure we have encountered an unexpectedly high incidence of abnormally high or low protein-bound iodine values (21). On clinical grounds none of these patients was suspected to have thyroid dysfunction. It is obvious that the existence of either thyroid hypo- or hyper-activity might well play an important if not a decisive role in the appearance of congestive symptoms and signs. This observation adds assurance to the clinical practice of treating cardiac patients, for a while at least, as if an element of thyrotoxicosis were present. The disadvantages or dangers of marked hypothyroidism must of course also be kept in mind.

Some comment is in order concerning the role of the central nervous system and the hypothalamus on thyroid activity. It is known that patients with organic lesions at the base of the brain tend to run low protein-bound iodine values (22). Whether or not these represent changes of the type seen in pituitary myxedema is not clear. Neither do we know anything about the TSH secretion in such subjects. In an attempt to answer some of the obvious questions we have been testing parahydroxypropioiphenone (23). This compound has been reported to suppress thyroid-stimulating hormone secretion; to date studies in this country have failed to confirm this finding (23, 24).

Finally, the advent of 11-oxygenated steroids and of ACTH has led to the observation that these substances also suppress circulating PBI and thyroxin (25, 26). This appears to be related to decreased production of thyroid hormone. Its importance during treatment with these agents has already been voiced by others (27), who have pointed out that the therapeutic efficiency of these agents may be adversely affected.

## IV. IODINE FRACTIONS DURING THE INGESTION OF INORGANIC IODIDES

The intake of as little as 0.2 to 0.4 grams of potassium iodide daily is associated with a slight but unequivocal increase in the serum precipitable iodine by the end of four weeks or earlier. At that time the upper limit of normal is usually reached or even exceeded (28). In such subjects the inorganic iodide level is considerably elevated, ranging between 100 to 300 gamma per cent. The use of an inorganic iodide in somewhat greater dosage, 0.6 grams daily, is associated with higher inorganic iodide values and an indisputable rise in the protein-bound iodine. Finally dramatic rises in the inorganic iodide up to as high as 22,000 gamma per cent, and in the protein-bound fraction up to 33 gamma per cent have been recorded in patients receiving massive daily doses of potassium iodide during sufficiently prolonged intervals (29).

Certain possibilities should be considered in discussing the mechanisms whereby these rises are induced. First the question as to the reality of the increase in protein-bound iodine can be promptly dismissed. These observations are not artefacts resulting from incomplete removal of inorganic iodide. Control studies have convincingly demonstrated that 14 washings of the precipitated serum with suitable portions of iodine-free water completely remove all inorganic iodide not bound to protein. This finding is confirmed by the excellent agreement in the analyses of samples washed both 14 and 18 times, indicating that all free inorganic iodide has been eluted. The persistence of high protein-bound iodine values following the cessation of iodide therapy and the return of the serum inorganic fraction of this halide to ordinary levels again supports the interpretation that the organic iodine fraction has indeed risen. It is conceivable that the observed increases in protein-bound iodine were mediated through the thyroid gland. The work of Taurog and Chaikoff in which the thyroxin content of rat glands rose ten-fold during iodide therapy makes such a mechanism plausible (30). Unfor-

tunately analyses of human thyroids during or after such treatment are not available. Serum thyroxin studies in the studies cited above (29) based on the relative solubilities of serum iodine fractions in butanol and a mixture of NaOH and  $\text{Na}_2\text{CO}_3$  provide a partial answer to this question. It is clearly evident from the studies cited that in no instance was the rise in protein-bound iodine accompanied by an increased thyroxin concentration. This certainly indicates that the non-thyroxin moiety of the protein-bound iodine has risen during iodide administration. Though it is conceivable that this rise was mediated through the thyroid gland, the alternative possibility however of an *in vivo* iodination of serum proteins must be kept in mind. Up to this time it has not been possible to reproduce these results *in vitro* by the incubation of serum or blood for relatively short intervals up to five days in length (31). These results pointing to a rise in the non-thyroxin protein-bound iodine while iodide is administered are in keeping with the clinical findings in such subjects. Hypermetabolic effects were conspicuous by their absence, and furthermore, such rises failed to correct the hypometabolism of myxedematous individuals.

These effects in humans have been reproduced in rats irrespective of the route of inorganic iodide therapy, i.e., either oral or parenteral iodide administration raised the non-thyroxin portion of the protein-bound iodine. On the other hand the subcutaneous injection of elemental iodine in propylene glycol increases to a modest degree the circulating thyroxin while inducing marked increments in the circulating protein-bound iodine (32, 33). These observations fit in very well with the convincing morphological and histological studies of Dvoskin which indicated without cavil that injected elemental iodine effectively replaced the secretion of the thyroid gland in influencing body growth, adrenal weight, and thyroid histology (34). Our own contribution to the problem unfortunately both answers and raises questions (33). Though goitrogens do influence the magnitude of the protein-bound and



thyroxin iodine change, we cannot at this point state the degree to which, if any, this represents influence over the extrathyroidal formation of thyroxin. More precise answers should become available in studying animals which are truly athyroidal established as such either by thyroidectomy or by serial and detailed histological examination, or by  $I^{131}$  and circulating PBI studies. Finally, it should be pointed out that if such extrathyroidal syntheses of materials mimicking thyroid hormone are occurring, and the bulk of evidence indicates this is indeed so, the question immediately arises as to whether or not this is mediated through enzyme systems related to those functional in the thyroid itself. A partial answer is available from our rat studies in which rises of protein-bound iodine were influenced in part by propylthiouracil (33).

#### V. STUDIES OF PROTEIN-BOUND IODINE DURING AND FOLLOWING INGESTION OF DESICCATED THYROID

Certain thyroid feeding experiments have further defined the response of endogenous thyroid hormone production during supplemental or replacement therapy. Tracer  $I^{131}$  studies have indicated that ingestion of desiccated thyroid depresses the trapping of iodide by the thyroid gland (35). Experiments in this laboratory indicate that usually more than 3 grains of thyroid have to be given each day to healthy young adults before the circulating protein-bound iodine rises above pre-treatment levels (36). Upon withdrawal of the medication, and irrespective of whether or not a change did occur during therapy, the protein-bound iodine dropped abruptly below values characteristic of the individual under study. This again points to suppression of the endogenous production of thyroid hormone, and is in keeping with similar observations made in patients receiving up to 25 grains of desiccated thyroid daily (37). These findings have a therapeutic implication since they suggest that thyroid administration to euthyroid subjects in amounts such as 1 or 2 grains merely

replaces an equivalent endogenous production of hormone. The net effect is a zero change. This would of course not hold in hypothyroid individuals nor would it apply to dosages in excess of the daily output by the gland.

#### SUMMARY AND CONCLUSIONS

In recapitulating the known facts about the serum iodine fractions in healthy adults it can be stated with fair assurance that the protein-bound and thyroxin moieties remain relatively constant during intervals up to at least several months in length. Menstruation or ovulation, or both, appear to be associated with minor fluctuations. Uncomplicated pregnancy is characterized by early rises in protein-bound iodine and thyroxin; in many patients who abort spontaneously such rises have failed to occur. Abnormally low protein-bound iodine values are frequently present during the first year postpartum, suggesting that pregnancy may be followed by a period of hypothyroidism.

Studies in children indicate that at birth protein-bound iodine levels are high, reflecting in part the elevation present in the mother and in part the great need for thyroid hormone during this very active phase of growth and development. The administration of thyroid to premature infants does not accelerate their maturation but does show that compared to adults, on a per kilogram of body weight basis, larger amounts of desiccated thyroid must be given before symptoms of thyrotoxicosis are elicited. In older children comparable levels of protein-bound iodine have been found in diabetic and in non-diabetic children. At the approximate time of pubescence the iodine value drops in both groups.

In addition to its usefulness in setting up correlations of the type cited above the protein-bound iodine levels offer a precise way of identifying hyper- and hypo-thyroidism. The feeding of inorganic iodine in large doses or for prolonged periods of time, the ingestion of desiccated thyroid, and the administration of organic sub-

stances such as lipiodol, produce elevations in protein-bound iodine not attributable to hyperthyroidism. A combination of analytic and clinical techniques will usually serve to identify such high values. On the other hand alterations in serum proteins or withdrawal of desiccated thyroid medication may be accompanied by low protein-bound iodine values in the absence of hypothyroidism.

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## EXCISION OF AORTIC ANEURYSMS\*

HENRY T. BAHNSON, M.D.†

Many forms of surgical treatment have been applied to aneurysm of the aorta, but those which have been of more than passing interest may be grouped under the following headings. *Ligation* is applicable only to aneurysms in the distal aorta and there is considerable risk to both patient and his extremities. *Wiring*, particularly with Dr. Blakemore's method of electrothermic coagulation, has given good results, but in our hands a number of aneurysms so treated have subsequently progressed in size. So called "*cellophane wrapping*" has been widely used, but even when the material is coated with dicetyl phosphate the results are inconstant. *Endoaneurysmorrhaphy* is an established method on peripheral arteries but has been rarely used on the aorta. There are a few scattered reports of *excision* of aortic aneurysms, but except for Tuffier in the early 1900's and recently Cooley and De Bakey most of these authors have not advocated excision. I shall attempt to show that excision is feasible in all regions of the aorta and that in many instances it is the treatment of choice.

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† From the Department of Surgery, The Johns Hopkins University School of Medicine and The Johns Hopkins Hospital. Aided by a grant from U. S. Public Health Service, National Institutes of Health, Bethesda, Maryland.

The principle causes of aortic aneurysms are two, namely arteriosclerosis and syphilis (Fig. 1). There are occasional aneurysms caused by trauma or bacterial infection but in general these resemble aneurysms due to syphilis. Syphilitic aneurysms usually occur in the thorax and almost always above the renal arteries. They are sacular, tend to occur in younger individuals and are often associated with pain or erosion of bone. The prognosis for such patients is quite poor, as only 18 of 633 patients reported by Kampmeier lived for more than two years. Most patients with aneurysms due to syphilis will die within six to nine months from the onset of symptoms. Arteriosclerotic aneurysms are usually fusiform, occur in older patients, below the renal arteries, and are usually not associated with significant pain or erosion of bone. About one out of five patients seen with arteriosclerotic aneurysms will die within a year of rupture of the aneurysm and over half of them will die within eight years.

Both types of aneurysms are amenable to excision. Aneurysms due to syphilis are usually localized ruptures with a narrow mouth. If this point of maximum weakness can be removed, the remaining aorta may be functionally adequate. On the other hand the position of arteriosclerotic aneurysms in the terminal aorta allows occlusion

of the aorta for excision and aneurysmorrhaphy or substitution by a homograft.

The following cases illustrate our method of handling aneurysms in various regions of the aorta.

Aneurysms on the convexity of the ascending aorta are particularly accessible to surgical attack. However, the mouth of the aneurysm must be isolated and occluded since the aorta in this

in his arms and neck but not in the lower extremities. Dullness and a definite pulsation were present in the first two intercostal spaces to the right of the sternum. There was no evidence of aortic insufficiency or cardiac failure.

On roentgenography a mass in the right mediastinum was noted which pulsated and pushed the lower trachea and right main bronchus posteriorly. The esophagus was deviated to the left at this level. Angiocardiography showed marked compression and partial obstruction of the superior vena cava which limited opacification of the aorta. A small polyethylene catheter was inserted percutaneously into a femoral artery and advanced to the ascending aorta. Injected contrast medium showed a saccular aneurysm on the right lateral wall of the mid-portion of the ascending aorta. There was no evidence of clot.

The right thoracic cavity was entered through the fourth intercostal space and the sternum was split upwards in the midline. The aneurysm was inside the pericardium. The mouth of the aneurysm was dissected free without disturbing intimate attachments to the right pulmonary artery and superior vena cava. The mouth was occluded with a straight intestinal clamp, the ends of which were compressed manually as the aneurysm was opened and three curved Potts coarctation clamps were applied to the cut edges. The opening was closed with two rows of interrupted fine silk sutures. The patient made an uneventful recovery.

The first patient we treated by excision was a 56 year old man referred to Johns Hopkins because of shortness of breath of four months duration. He was unable to work and was dyspneic at rest. There was evidence of syphilis. On tracheoscopy an extrinsic mass almost occluded the trachea by pressure from the left in the region of the aorta. Laminography demonstrated this striking compression. Angiocardiography showed a small but strategically located aneurysm on the dome of the aortic arch. We opened the chest widely, splitting the second left interspace and the sternum (Fig. 2, a). The aneurysm

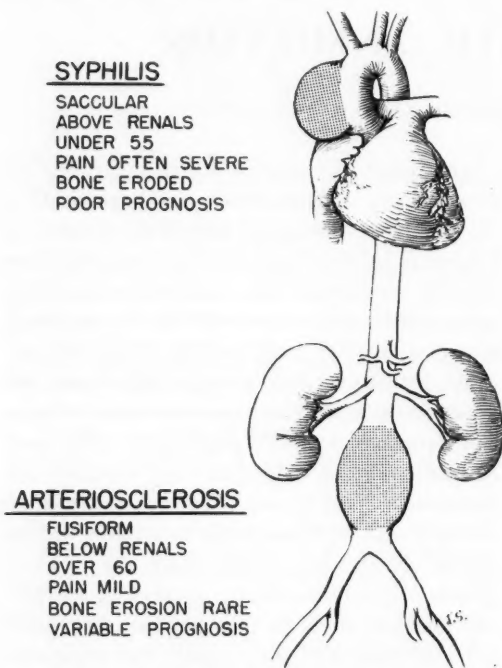


FIG. 1. Diagram illustrating two principal types of aortic aneurysms with their clinical characteristics.

region cannot be obstructed for more than a very short period.

Such a case is illustrated by a 51 year old colored male in whom the diagnosis of syphilitic aneurysm was made in 1951. At that time he had dyspnea paroxysmally at night, dyspnea on exertion and substernal pain which was worse after eating and often kept him awake at night. These symptoms became more pronounced, and he lost 21 pounds in the year preceding admission.

On examination there was venous distention



arose from the medial portion of the aortic arch behind the left common carotid artery just proximal to the origin of the left subclavian. The left subclavian artery was ligated and a clamp placed across the base of the aneurysm and the aortic arch (Fig. 2, b). The aneurysm was excised and the base of it closed without difficulty by numerous fine sutures. In removing the sac,

3, c). He was ready to return to work two months later, and at last report, six months after opera-

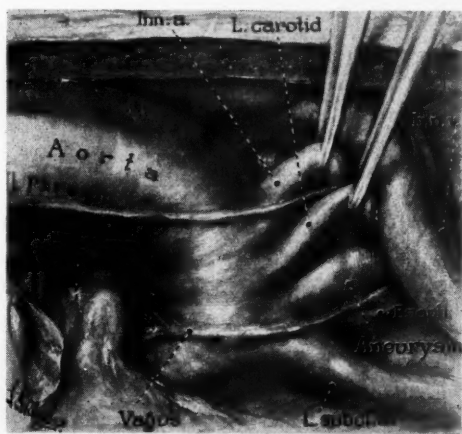


FIG. 2a

FIG. 2. A 56 year old man was seen because of progressive dyspnea. Marked compression of the trachea was demonstrated in the region of the aorta. The aneurysm was excised from the aortic arch, and the patient is well.

a. Exposure was through the second left interspace and split sternum. The aneurysm arose from the medial side of the aortic arch behind the left common carotid and subclavian arteries.

b. The left subclavian was ligated, a clamp placed across the base of the aneurysm and the aortic arch, and the aneurysm was excised. Closure was with numerous fine silk sutures.

c. Visualization of the trachea and esophagus 2 weeks after operation shows normal position and lumen of both.

which incidentally is a must to prevent subsequent infection and rupture, the wall of the sac was fused to the trachea. Flattened cartilages were in direct contact with blood and clot. Apparently rupture had been imminent. Tracheotomy was necessary after operation to aspirate the profuse bronchial secretions. A bronchogram and esophagram taken two weeks after operation just prior to removal of the tracheotomy tube showed normal position of both structures (Fig.

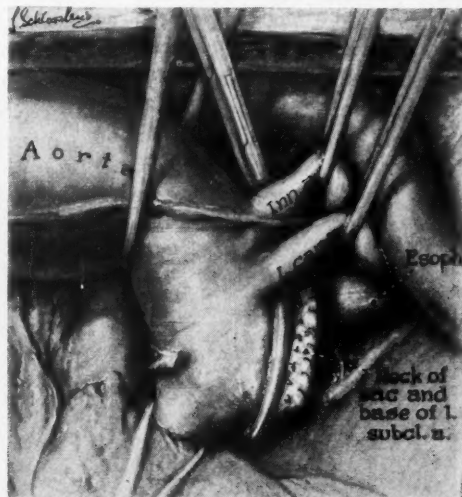


FIG. 2b



FIG. 2c

tion, he was well, although in the Maryland House of Correction.

One of our patients was a 49 year old registered nurse who sustained an injury to her chest in an automobile accident in October 1950. After

regaining consciousness she was dyspneic and had a sense of painful compression in the mid-sternal region. Hoarseness was soon noted. At no time could a blood pressure measurement be obtained in her left arm. Roentgenograms at that time showed no intrathoracic abnormality, but a month later a pulsating mass was discovered in the left upper mediastinum. She was referred

turned to normal but four months later hoarseness reappeared. Later pain reappeared in the upper chest. She returned to the hospital in September, 1952, for reevaluation. Repeat angiocardiology and chest film showed little if any change in the configuration of the aneurysm, and operation was thought advisable. At operation the aorta and vessels arising from the arch

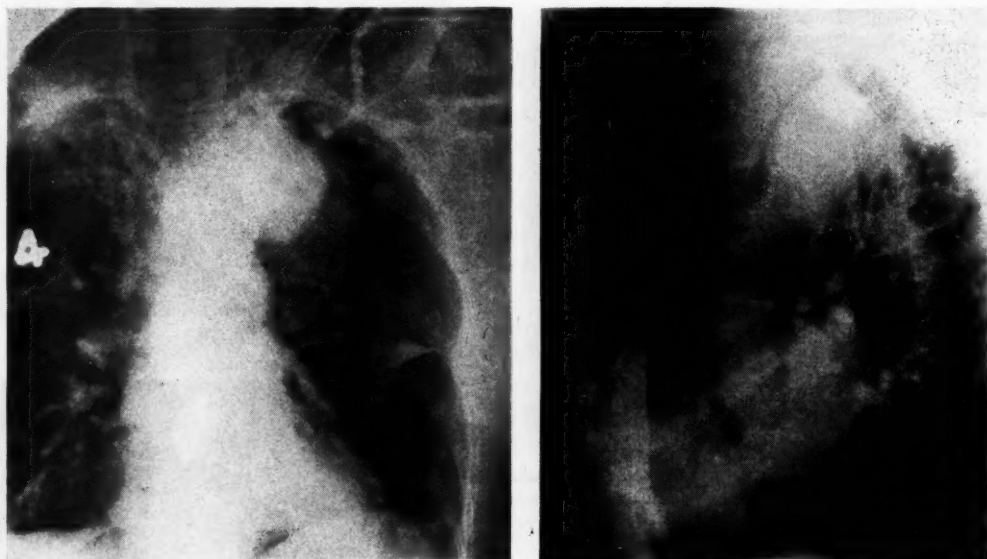


FIG. 3a

FIG. 3. A 49 year old nurse sustained an injury to her chest which resulted in an aneurysm in the terminal portion of the aortic arch. There was temporary improvement following "cellophane wrapping," but later reactivation occurred and excision was carried out successfully.

- a. Antero-posterior and lateral angiocardigrams showing the saccular aneurysm of the terminal portion of the aortic arch.
- b. Operative exposure shows the aneurysm of the distal aortic arch with the left subclavian artery arising from the aneurysm itself (upper drawing). The lower drawing shows the aneurysm opened and closure of the narrow mouth begun. The aorta was occluded proximal to the left common carotid artery and distal to the aneurysm for 7 minutes.

to Dr. H. William Scott at Johns Hopkins in June 1951. There was a significantly lower blood pressure in the left arm than the right. There was no evidence of syphilis. Angiocardiology showed a saccular aneurysm at the terminal portion of the aortic arch with considerable clot in the wall (Fig. 3, a). Dr. Scott wrapped the lateral surface of the aneurysm with reactive polythene film. Convalescence was uneventful and she became asymptomatic. Her voice re-

were isolated (Fig. 3, b). The aneurysm arose from the terminal portion of the aortic arch. The left subclavian arose from the aneurysm itself. The aortic arch just proximal to the left common carotid artery and the upper thoracic aorta were cross-clamped for seven minutes during which time the aneurysm was opened. A discrete circumferential tear was seen in the aorta at the site of origin of the left subclavian artery. The anterior portion of the opening was easily sutured



from inside the aneurysm. Because of beginning hypotension and cardiac irregularity cross clamping of the aorta was discontinued before the entire opening could be sutured. The hole in the

back following an automobile accident was seen a year after the injury when a pulsating mass appeared in the left costo-vertebral angle. Aortography by Dr. Willard Goodwin showed a

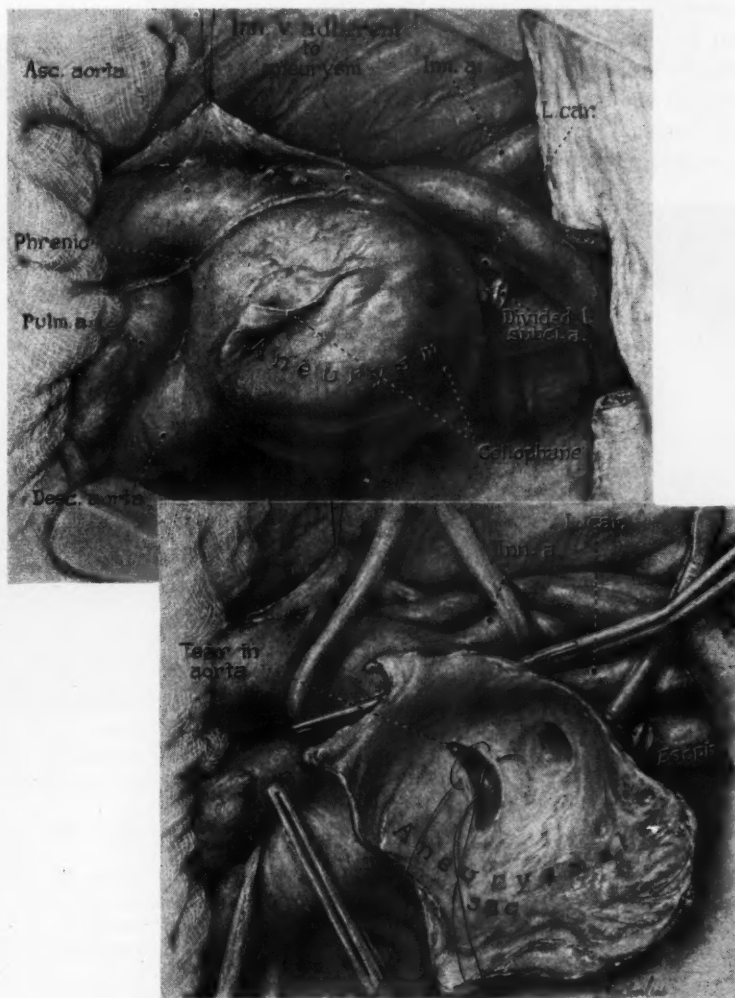


FIG. 3b

aorta was controlled by manual compression against the spine and aorta while the aneurysm was completely mobilized and the opening closed. She has done well since operation.

A 45 year old colored male who had pain in his

bilobate upper abdominal aneurysm (Fig. 4, a). Wiring and electrothermic coagulation were performed with what we thought was a satisfactory result. After a brief remission the mass enlarged and when readmitted about a year ago there

was a large mass in the left flank which encroached on the costal margin above and the iliac crest below (Fig. 4, b). Aortography showed considerable enlargement of the lumen of the aneurysm and an additional 300 feet of stainless steel wire was inserted. There was considerable relief of pain but the mass seemed to enlarge. Excision was thought indicated. Wide exposure was obtained through the bed of the tenth rib and a thoraco-abdominal incision. The abdominal viscera were displaced forward and to

aneurysm was excised and the aorta reconstructed from aneurysmal wall. Vertebral bodies, spinal nerves and in one area dura were exposed to blood in the aneurysmal lumen. Just prior to discharge an aortogram showed a tortuous aorta



FIG. 4a



FIG. 4b

FIG. 4. A 45 year old man was seen with a painful upper abdominal aneurysm. Wiring and electrothermic coagulation relieved the pain and pulsation ceased. The mass later enlarged and symptoms recurred. The aneurysm was excised and aorta reconstructed from wall adjacent to the mouth. The patient is now well after a long convalescence.

a. Initial aortogram shows a bilobate upper abdominal aortic aneurysm. This was inactivated by wiring and electrothermic coagulation, but the aneurysm subsequently enlarged and extended into the pelvis.

b. At the time of second hospital admission the mass encroached on costal margin above and iliac crest below.

c. Thoraco-abdominal exposure through the bed of the tenth rib. Abdominal aorta and viscera were displaced forward by the large aneurysm. Visceral branches and the aorta proximal and distal to the aneurysm were occluded for 37 minutes while the aneurysm was excised and the aorta reconstructed. Vertebral bodies, spinal nerves and dura were exposed to blood in the aneurysmal lumen.

d. Aortogram 1 month after excision shows a tortuous aorta with a relatively normal lumen.

the right (Fig. 4, c). The mouth of the aneurysm extended from just posterior to the origin of the celiac axis down to the level of the renal arteries, a distance of 12 to 15 centimeters. The vessels and aorta proximally and distally were isolated and occluded for 37 minutes while the

but a relatively normal lumen (Fig. 4, d). He had a long convalescence but when recently seen, over a year after operation, he was well.

As an example of an arteriosclerotic aneurysm a 67 year old man noted a pulsating abdominal mass over a year before admission when he was



FIG. 4c



FIG. 4d



FIG. 5a. (See page 544 for legend.)

under observation for a probable coronary thrombosis. The mass was occasionally tender but otherwise asymptomatic. The patient was

area and the opacified lumen represented what we thought was localized clot several centimeters thick (Fig. 5, a). At laparotomy the lower ab-

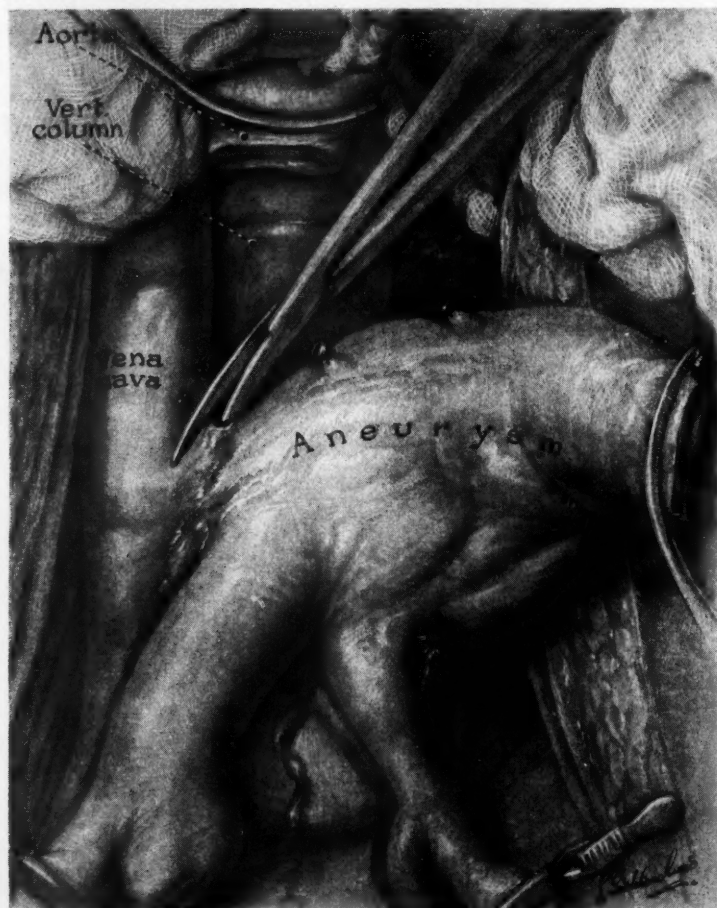


FIG. 5b

FIG. 5. A 67 year old man had an aneurysm of the distal abdominal aorta due to arteriosclerosis. It was excised and replaced by a homograft of the same region. The patient is now well.

- a. Aortogram shows the irregular lower aortic lumen displaced to the right and anteriorly by localized clot.
- b. The aneurysm was exposed through a long paramedian incision. The aorta and iliac arteries were occluded, and the aneurysm was excised.
- c. A homograft has been sutured in place between the aorta and external iliac arteries.

confident that it had recently enlarged. Aortography showed a slightly dilated but smooth lumen of the upper abdominal aorta. The lower abdominal aorta however was irregular. Calcium could be seen in one region and between this

dominal aorta from just below the renal to the external iliac arteries was involved. The entire area was excised and replaced with a graft of the same region preserved in tissue culture medium (Fig. 5, b and c). There was oliguria

during the early post-operative period but he subsequently made an uneventful convalescence.

In the past 15 months we have operated upon

the right internal carotid artery, and the other died from inadvertent occlusion of the left common carotid during planned occlusion of the



FIG. 5c

20 patients with the intention of excising the aneurysm. Five of these patients died during or shortly after the operation. Of the 5 two were due to aneurysms on the ascending aorta. One of these died from a clot which was dislodged into

innominate. In this the incision and exposure was limited because the aneurysm had perforated the chest wall and we were hesitant to split the sternum as has been done in other patients with aneurysms in this location. A third patient who



did not survive had a compound fusiform and saccular aneurysm of the entire aortic arch; we would not explore this patient at the present but at that time hoped that a saccular aneurysm in the concavity of the aortic arch could be excised with relief of his pain and respiratory obstruction. The fourth patient was a young white man without syphilis who had a large aneurysm involving the entire thoracic and upper abdominal aorta. He died the day following the excision and replacement by two thoracic aortic homografts sewed end-to-end. There was no explanation of his death other than surgical shock. The final death was for an arteriosclerotic aneurysm in a patient with an acute rupture who was in shock when the operation was begun and died before the aneurysm was excised. Of the 15 successful operations 3 patients are still in the hospital convalescing from recent operation. The other 12 are well and essentially symptom free. This group includes the patients reported in detail, one patient with an innominate and aortic aneurysm, another with an aneurysm on the ascending aorta, and one with an aneurysm on the upper thoracic aorta.

In addition to the last patient reported in detail we have successfully operated upon seven others with arteriosclerotic aneurysm in the terminal aorta. One of these was treated by aneurysmorrhaphy, a method which is less satisfactory than aortic homografting. One of the seven homografts was preserved in tissue culture medium. The others were frozen and dried. We are indebted to Dr. Russell Fisher and his staff for helping us procure most of these homografts. Two of the frozen dried grafts which were used

were obtained from the Tissue Bank at the Naval Medical School, Naval Medical Center, Bethesda, Maryland. In four of the homografts, anastomoses were between the aorta and both iliac arteries. In one of these, done by Dr. David Sabiston, the left iliac artery was anastomosed to the side of the thoracic aortic graft; in the others bifurcation grafts were used. Three patients had excision of the terminal aorta with substitution of a segment of aorta without the branches. The only significant permanent complication in this series occurred in a patient who is still in the hospital. Occlusion of the artery in the thigh caused gangrene which required amputation of the leg. Significant oliguria occurred in two patients in the post-operative period. The others have been surprisingly free of post-operative difficulties.

In conclusion excision of aortic aneurysms has been successfully performed in all sections of the aorta and appears to be the method of choice in many instances.

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#### MARYLAND RADIOLOGICAL SOCIETY

At the initial meeting of the Maryland Radiological Society, the following officers were elected for the coming year: *Chairman*, Richard B. Hanchett, Baltimore; *Vice-Chairman*, E. T. Campbell, Hagerstown; *Secretary-Treasurer*, H. Leonard Warren, Baltimore.



## THE INDICATIONS FOR AND THE RESULTS OF SURGERY FOR MITRAL AND AORTIC STENOSIS\*

ROBERT P. GLOVER, M.D.†

The recent "Renaissance of Cardiac Surgery" has introduced new hope and assurance both to the medical profession and to the patient suffering from the cicatricial and disastrous end results of rheumatic fever. The demonstration in the past decade that the interior of the heart could be successfully invaded at will for the reconstruction of stenotic valvular disease inaugurated this new era of surgical achievement.<sup>1-3</sup> Surgery, almost from its inception, has had as one of its primary objectives the relief of "obstruction and stricture" no matter where the offending block lay within the systems of the human body. It was logical, therefore, that with the application of basic surgical principles to cardiac disease initial attention and effort be directed toward the relief of "obstructed and strictured" valves.

Any consideration of valvular lesions, surgical or otherwise, must at once take into account the nature of their pathologic change. For reasons not entirely clear the valves within the heart predominantly undergo malformation according to their location within the vascular pump. Thus, the valves in the right heart (tricuspid and pulmonary) are primarily the seat of congenital deformities.<sup>4-6</sup> On the other hand, the mitral and aortic valves in the left heart are damaged by superimposed acquired disease, usually rheumatic in origin. Whereas, prior to 1950, the congenital anomaly monopolized the surgeon's attention, today by far the greatest field of cardiac surgical endeavor is concerned with the problems of acquired disease. Already the stage

of initial conjecture and speculation as to the proper application of newly devised intracardiac surgical techniques is passing. Experience to date has shown that surgical intervention at the proper time and in intelligently selected cases can be considered a major adjunct in the overall care of the rheumatic heart crippled by valvular stenosis.<sup>6-10</sup>

### MITRAL STENOSIS

#### *Pathophysiology*

In rheumatic disease the mitral valve develops numerous minute inflammatory verrucae in a row along the line of closure of the valve. With continuing rheumatic activity and attempts at healing over the course of years there is a gradual development of fibrosis, thickening, and narrowing of the valve leaflets as their cusp margins become adherent at the angles (commissures). This scarring may be limited in extent to resemble a purse-string puckering at the valve orifice with minimal involvement of the valve leaflets themselves, which remain quite pliable and of the consistency of kid glove skin. In other instances, the periorificial induration involves one-fourth to one-half of the valve cone leaving a correspondingly smaller margin of flexible tissue about the base at the A-V ring. In far advanced disease the whole valve may become rigid and completely inflexible—a hard, ovoid plaque surrounding a tiny fish-mouth slit. Calcium may be present at any stage as flecks, localized infiltration or, rarely, almost completely replacing valve tissue. Thus, as stenosis is produced, pronounced resistance to the passage of blood from the left atrium into the left ventricle ensues. Since the egress of blood from the left atrium is impaired, increased pressure within and considerable dilatation of this chamber re-

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sults. The high intra-auricular pressure is transmitted to the entire pulmonary vascular system and thence to the right side of the heart. A chronic pulmonary hypertension results with nocturnal or exertional congestion (dyspnea), rupture of pulmonary capillaries (hemoptysis), and failure of the right side of the heart (hepatomegaly, ascites, and peripheral edema).

Once this progressive pattern becomes heralded by the onset of fatigue and exertional dyspnea the ultimate outcome for the patient is in time invariably unfavorable. At this point the treatment of the physician will be directed toward the support of a myocardium which is vainly attempting to maintain an adequate systemic circulation in the face of an unrelenting mechanical stricture. The fort can be held temporarily but eventually under such circumstances both the physician and the myocardium are fighting a losing battle. It is paramount to recognize that the earliest onset of symptoms bespeaks a failing myocardial and pulmonary vascular reserve as the result of *already long-standing structural valvular stenosis*. To repeat, with the onset of symptoms the valvular lesion for the most part has already reached its ultimate cicatrix and progression of the patient's disability is one of symptomatic breakdown and disintegration, not of increasing structural stenosis within the valve itself. The therapeutic conclusion is obvious. The stenotic valve must be opened at the earliest suggestion of the above described obstructive phenomena if one is to avoid the inevitable progression of enlarging cardiac chambers (left atrium, right ventricle, right atrium), pulmonary edema, recurrent hemoptysis, auricular fibrillation, embolic episodes, and chronic congestive (right heart) failure.

#### *Clinical Classification*

The following classification has been prepared to provide a clinical, functional yardstick paralleling the progressive pathophysiologic changes as outlined above.

#### *Stages of Mitral Stenosis*

- I. Asymptomatic
- II. Statically incapacitating
- III. Progressively incapacitating
- IV. Terminally incapacitating
- V. Irretrievable

*Stage one* includes those with the auscultatory findings of mitral stenosis but who as yet have no symptoms. Cases in *stage two* have progressed to the point at which dyspnea and fatigue under physical stress have developed but the patient, with or without medication, living within his own limitations remains on an even plateau. *Stage three*, inevitably the largest group and one encompassing many variables, includes those who, despite the best medical therapy, are slowly losing ground, i.e., are faced with increasing reliance upon diuretics, daily bed rest, etc. In *stage four*, terminally incapacitating, are those patients in whom there is constant evidence of congestive failure even with reasonably limited physical activity. Most of these can be rendered relatively free of their accumulating tissue fluid only by the strictest of medical regimens, including constant rest in bed. Even then, not infrequently, one is unable completely to reduce hepatic congestion. A certain small percentage of those in this group will ultimately prove after surgery to have been in *stage five* and to have had irreversible pulmonary arteriolar changes. As yet, it has been impossible routinely to separate patients in these two stages by clinical and physiologic methods now available hence we reserve *stage five* to classify those who, despite a technically adequate commissurotomy, receive no improvement because of irreversible cardiopulmonary pathologic change.

#### *Surgical Objective*

It is sufficient to state here that mitral commissurotomy is a procedure in which the individual anatomic leaflets of the stenotic valve are separated. By incising the angles or commissures of the mitral slit, its obstructing effect can be overcome and a considerable degree of valve function can be reestablished. It is to be

noted that no valve tissue is removed, thus allowing the liberated though thickened and deformed valve leaflets to open during ventricular diastole and approximate during ventricular systole without the production of additional significant regurgitation (Fig. 1).

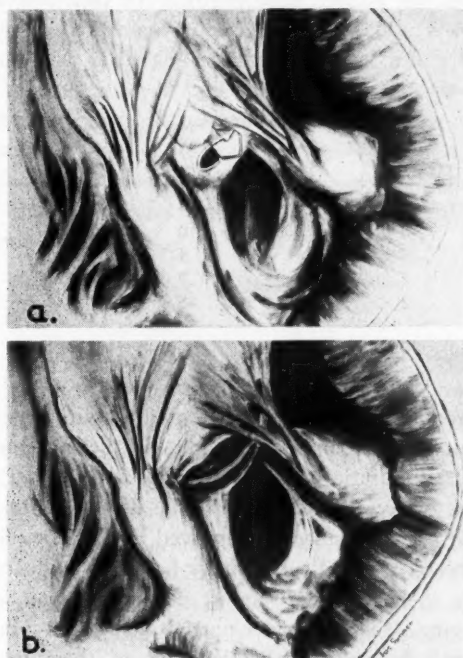


FIG. 1. A. Left ventricular view of the mitral valve cone in stenosis. The right index finger and Glover-Avery guillotine have been inserted through the left auricular appendage and properly placed for incision of the anterolateral commissure. B. Valve orifice after commissurotomy. Note the intact chordae tendineae insuring partial restoration of valve function with prevention of significant mitral insufficiency.

#### Indications for Commissurotomy

In the light of the foregoing presentation, the selection of patients for surgical intervention can be simply stated. The *ideal candidate* is the patient with pure mitral stenosis and beginning symptoms of cardiopulmonary dysfunction such as shortness of breath upon exertion. Fatigue out of all proportion to the patient's physical activity is frequently a prodromal or accompanying finding. The mere presence of a well

defined mitral diastolic murmur without accompanying symptoms is insufficient reason to suggest surgery, for the operation is not performed primarily because of the murmur but to improve the patient's functional status.

All other indications for commissurotomy are merely compromises from the ideal but for years to come an understanding of the more advanced but altogether salvageable states is essential. An outline under seven major categories will most succinctly serve this purpose:

1. History
  - a. Early cardiopulmonary dysfunction—ideal.
  - b. Marked dyspnea, hemoptysis, reversible failure—acceptable.
2. Age: Elastic range—physiologic rather than chronological. (Oldest age 61)
3. Valvular defect.
  - a. Pure mitral stenosis—ideal.
  - b. Associated mitral insufficiency and/or aortic valve lesion in presence of normal left ventricle—acceptable.
4. Roentgen findings.
  - a. Left atrium and right ventricle minimally enlarged—ideal.
  - b. Minimal left ventricle enlargement—questionable but acceptable.
5. Electrocardiogram.
  - a. Normal electrical axis or right ventricular strain—ideal.
  - b. Left axis shift—never acceptable.
  - c. Auricular fibrillation with controllable ventricular response—acceptable.
6. Functional capacity.
  - a. Stage 2 (statically incapacitating)—ideal.
  - b. Stage 3 (progressively incapacitating)—acceptable.
  - c. Stages 4 and 5 (debatable)—occasional good result.
7. Complicating factors.
 

<ol style="list-style-type: none"> <li>a. Arterial embolic episodes</li> <li>b. Recurrent hemoptysis</li> <li>c. Pregnancy</li> </ol>	}	Acceptable, and may be urgent.
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8. Contraindications.
  - a. Acute rheumatic fever.
  - b. Subacute bacterial endocarditis until controlled.
  - c. Associated marked mitral and aortic insufficiency with all cardiac chambers enlarged.

#### Analysis of Cases

Five hundred mitral commissurotomies have been performed by the author and his partner,

Dr. Thomas J. E. O'Neill: The operative mortality rate for the entire series has been 5.9%. A statistical breakdown of the first 164 of these cases in whom complete data to the present day is available (2 to 4½ years) can be seen in table 1. It will be noted that no cases have been attempted in Stage 1, and this is as it should be in

salvage on a calculated risk basis and should be subjected to surgery only with the patient's full knowledge and understanding of the risks involved.

The results, as graphically portrayed in Figure 2, are self-explanatory. Excellent (49%) means that the patient has been restored to a normal,

TABLE 1  
Mitral Stenosis

STAGE OF STENOSIS	NO. CASES	DEATHS		MORTALITY %	
		Operative	Late	Operative	Late
I. Asymptomatic.....	0	0	0	0	0
II. Statically Incapacitating.....	14	0	0	0	0
III. Progressively Incapacitating.....	105	5	1	4.7	.9
IV. Terminally Incapacitating.....	32	3	0	9.3	0
V. Irretrievable.....	13	2	8	15.3	61.5
Total.....	164	10	9	6.1	5.5

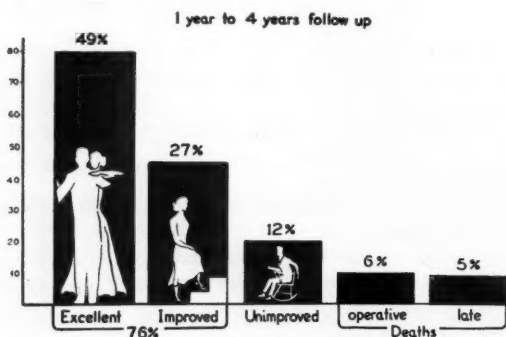


FIG. 2. Results of Mitral Commissurotomy

the present state of such a program. The operation should not be undertaken on the basis of a murmur alone, but for the relief of the patient's functional deterioration of which the murmur is but an auscultatory incident. However, many more than have been in the past should be referred for surgery when in Stage 2 for by this time it is obvious that myocardial strain and ultimate breakdown is at hand. Stage 3 represents an imperative indication beyond which the patient under good care should not be permitted to progress. Stages 4 and 5, frequently undistinguishable clinically, represent pure

productive life, enjoying normal activities without obvious limitation. A low salt diet and mercurial diuretics are no longer necessary. Most of these have dispensed with the use of digitalis. 27% are classified as improved in that they are objectively and subjectively better as evidenced by their return to almost normal life and activity. Most are under partial medication (digitalis) because of the demands of their more greatly enlarged sphere of activity. A few, terminally incapacitated before surgery, have now become so amenable to therapy that reasonable activity requires digitalization only. The range of improvement in this group varies widely with the condition of the valve found at surgery and the degree to which valve function could be restored. Their progressively downhill course has been abruptly terminated or reversed, some to regain a high level of efficiency and others to remain on an improved plateau.

Twenty patients (12%) have been essentially unimproved. Multivalvular disease and significant mitral insufficiency, the extent of which was not entirely appreciated until valve exploration, were the major causes for the unsatisfactory outcome.



The operative mortality in the 164 cases closely parallels the mortality in the entire series and was 6.1%. As might be expected, the deaths occurred in the more advanced stages of the disease (Table 1), and were due primarily to the unyielding nature of the valvular pathology or to improper technique as applied to the pathologic state present (operative, anesthetic and diagnostic). The late deaths were all occasioned by the presence of pre- and postoperative irreversible cardiovalvular and pulmonary-hepatic vascular (and parenchymal) disease which remained unaffected by the mere relief of mechanical obstruction and progressed to their normal termination.

#### AORTIC STENOSIS

The problems associated with the surgical alleviation of aortic stenosis have to date presented far greater difficulties than any of the other stenotic valvular lesions. There are a number of reasons for this.

*Anatomically*, the valve is the least accessible to exploration, for it lies centrally placed within the confines of the heart. In aortic stenosis, the left ventricular myocardium is greatly hypertrophied, which, together with the tremendous vascular pressure generated within its outflow tract, prevent the use of a subaortic transmyocardial incision for the insertion of finger or instrument similar to that used in pulmonary valvulotomy, because of the ensuing uncontrollable hemorrhage. The valve cannot be effectively reached through an auricular appendage. The position of the ostia of the coronary vessels lying directly behind the valve leaflets makes direct reconstructive surgery hazardous.

*Pathologically*, there is an early fusion of the three valve cusps, the margins of which become thickened, rolled, eburnated and infiltrated with calcium very early in the course of the disease. This is due, in part, to the disease itself but more particularly to the location of this valve in the course of the blood stream. The valve lies in a confined space of small diameter and is the recipient of the full head of vascular pressure

from the strongest of contracting cardiac chambers—the left ventricle. The factor of *vascular trauma* is, therefore, of great importance for, following initial fibrotic agglutination of the cusp margins, there is a constant and steady deposition of traumatic elements such as fibrin and calcium from which there can be no relief. Thus, calcification develops early and as the leaflets are relatively small in surface area, almost complete immobility and fixation may be rapidly evident. Some flexibility of the cusps at their attachment to the aortic ring may remain for a time but this is less prominent than that seen in the stenotic mitral valve. This factor of *trauma* no doubt partially explains the fact that the antero-medial leaflet of the mitral valve (also called the aortic or septal leaflet) is the seat of the greatest calcification in mitral stenosis, for it lies nearest the aortic ring in the high pressure left ventricular outflow tract and also received the brunt of the vascular ejection at the time of left ventricular contraction. Therefore, from a pathologic standpoint, surgical intervention in aortic stenosis, to effectively restore a measure of normal valve leaflet motion, must be carried out before extremes of the above described process are reached. To wait longer merely compounds the likelihood of surgical failure, for in consequence of the valvular change the left ventricular myocardium hypertrophies and dilates, in due time to wear out and become incapable of effective action. As the left ventricle is the cornerstone of the heart and circulation its failure predicates irremedial disaster. This is in contrast to the findings in mitral stenosis, where, despite profound pathological and clinical changes even to the point of prolonged congestive failure and invalidism, the left ventricle retains its indispensable function—actual, compensatory and recuperative—almost to the end.

*Physiologically*, because of the jet-like ejection of relatively small amounts of blood with each left ventricular contraction, the systolic blood pressure may not reach high levels although it is perhaps sustained and maintained longer than usual. Diastolic pressures, however, are high so

that the pulse pressure is small and coronary artery filling, now under greater stress because of the demands of an increased myocardial mass, may be inadequate. As such, these patients are

lem has been previously reported.<sup>11</sup> Briefly, the present operative approach calls for a left lateral thoracotomy, the introduction of a dilating instrument through a relatively avascular area of

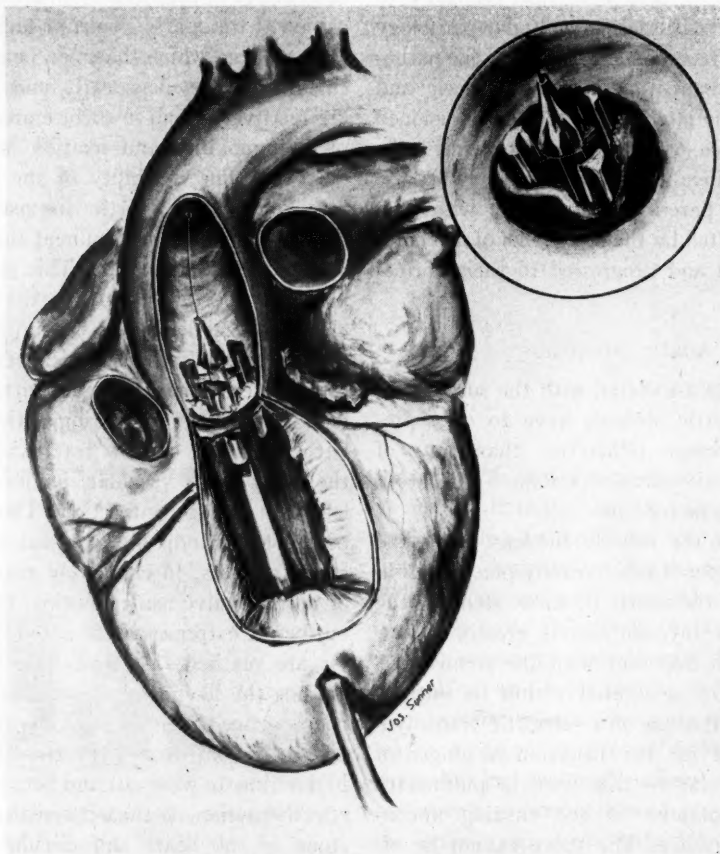


FIG. 3. The aortic dilator inserted through the myocardium of the left ventricle near its apex. This instrument is guided through the aortic valve by threading it over a fine olive-tipped wire previously placed through the valve. *Inset.* The three flanges of the dilating head separating the commissures. The self-rotating head settles automatically into the V-shaped commissures of the fused aortic cusps.

often precarious anesthetic risks, to say nothing of the superimposed danger of surgery.

#### *Surgical Objective*

Despite these apparent roadblocks, considerable progress has been made. The experimental work which has been undertaken in an effort to find a satisfactory surgical solution to this prob-

the left ventricle near its apex and lateral to the left descending coronary artery. The dilator is directed through the valve by passing it over a previously placed wire guide, the olivary tip of which can be felt in the aorta prior to the insertion of the larger instrument through the myocardium. The dilating head opens in triangular fashion causing the fused commissures of



the stenotic valve to separate, partially relieving the stenotic obstruction and restoring a measure of valve action (Fig. 3).

### *Clinical Classification*

The necessity for translating the present rationale of surgery for aortic stenosis into reasonable, practical language has called for the development of a classification for the recognition of clinical states either applicable or unsuitable for surgery at the present time.

tightness, effort angina with its characteristic distribution bespeaks of inadequate coronary flow. With beginning left ventricular failure, Stage 4 is characterized by episodes of pulmonary congestion and edema at first rather easily controlled by medical measures but shortly to become refractory to even the most vigorous management. Stage 5 connotes impending disaster and an early demise for right heart failure with hepatomegaly, ascites and peripheral edema labels the heart picture as one of ir-

TABLE 2  
*Aortic Stenosis*

STAGES	RESULTS				
	No.	Excellent	Improved	Unimproved	Deaths
I. Asymptomatic—murmur only.....	0	—	—	—	—
II. Fatigue, Heart Consciousness, Palpitations.....	2	2	—	—	—
III. Syncope, Angina.....	10	2	6 (3*)	1	1
IV. Episodic Pulmonary Congestion.....	5	—	3	1	1 (1*)
V. Congestive Failure.....	5	—	—	—	5 (2*)
Totals.....	22	4	9	2	7

\* Combined mitral and aortic commissurotomies.

The recognizable clinical stages through which the average patient with aortic stenosis will pass are well known, although perhaps not readily defined in the mind of the physician in the light of surgical advances. Stage 1 is the period during which the typical systolic murmur develops before symptoms result. Stage 2 marks the onset of an ill-defined period where the patient becomes subjectively aware of his forceful heart action, palpitations develop, easy fatigue becomes noticeable and his attention may be called to the unusual, visible pulsations in his supraclavicular and suprasternal notches. Admittedly, this stage is not specifically characteristic of aortic stenosis per se, but when seen with an aortic systolic murmur the combination takes on added significance. Stage 3 ushers in the obvious period of progressive disability due to a diminishingly effective cardiac output as evidenced cerebrally by spells of dizziness and actual syncope. Myocardially, substernal discomfort,

retrievable damage not to be reversed by any regime—medical or surgical.

### *Results of Surgery (Table 2)*

Twenty-two cases have undergone aortic commissurotomy and six of these have been combined with a mitral commissurotomy because of concurrent bivalvular stenosis. One patient (Stage 2) done over three years ago remained completely asymptomatic for two years. During the past 12 months, although working steadily and living a normal life, he has noticed some minor precordial tightness with exercise after meals which had not been present during his first two postoperative years. This patient's aortic commissurotomy was performed with our original dilator<sup>2</sup> which did not have the strength or wide diameter opening of the present more refined instrument which may account for his present status. Clinically there has been no change in his cardiac findings.

The remaining twenty-one cases have been operated upon in the past eighteen months. The pertinent data can be seen at a glance in table 2. The overall mortality has been high which may have been influenced somewhat by the inclusion of cases with bivalvular surgery. The observation of paramount importance, however, is that cases done in the early symptomatic phase (12 cases—Stages 2 & 3) run a risk of only 8.3% and even including those in Stage 4, when pulmonary edema has entered the picture the mortality rate is a reasonable 11.7%. One has only to consider the seriousness of the disease and the utter hopelessness of its immediate prognosis to appreciate that such results are highly encouraging at this stage of our surgical programme. When true congestive failure has developed (Stage 5) it is doubtful whether much, if any, salvage can be either expected or obtained.

#### CONCLUSIONS

The natural course and progression of rheumatic stenotic valvular disease has dictated the necessity for the mechanical relief of its acquired stricture. Mitral and aortic commissurotomy have become major adjuncts in the overall and continuing care of victims so afflicted. Provided the patient is referred for surgical intervention early in the course of his symptomatic progression, such a patient can be restored to a high level of efficiency and enjoy a more normal life as a useful and productive citizen. The burden of selecting patients at an early stage rests squarely on the shoulders of the practicing phy-

sician and this responsibility can no longer be avoided or postponed.

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#### SOUTH DAKOTA STATE BOARD OF MEDICAL EXAMINERS

JOHN C. FOSTER\*

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\* John C. Foster, Executive Secretary.

## THE NON-OPERATIVE TREATMENT OF STRESS INCONTINENCE IN WOMEN\*

LAWRENCE R. WHARTON, M.D.†

Stress incontinence is a relatively common complaint among women. Gynecologists have also found it a difficult problem to understand and treat. This is true today in spite of the fact that during the past fifty years, this problem has been studied from every conceivable angle.

Among modern gynecologists, Dr. Howard A. Kelly was a pioneer in these investigations. In 1914, in conjunction with William Dumm, he published an article on this subject in which he attributed the incontinence to weakness of the internal sphincter of the urethra and described the operation, plication of the internal sphincter. At that time he stated that he had been studying the problem for many years and that he had been using this operation with a considerable degree of success for 10 years. That procedure has become one of the standard methods of treating this condition.

Since that time, interest in this syndrome appears to have increased with passing years. This has been directed generally into two main channels—study of the anatomic and physiologic factors that may have any bearing on the incontinence and the discovery and modification of new operations devised to correct it. Needless to say, an immense amount of time and energy has been expended in these efforts.

In spite of all of this, as Berkow stated in February, 1953, "the physiological mechanism of urinary control in the female has remained obscure" and most of us will agree with the conclusion of the London conference of 1950, quoted by Berkow, that the "exact mechanism of stress incontinence is not completely known."

A similar uncertainty marks the surgical efforts

to cure this condition. For many years, the Kelly plication of the vesical sphincter remained the operation of choice. It is still the author's preference, under ordinary conditions. In 1947, TeLinde published a follow-up study of 249 cases in which this procedure had been performed in the decade preceding 1947. He reported that 90.3 per cent had been cured, 5 per cent improved, 3.5 per cent failed and 1.2 per cent were not traced. This report is a little more favorable than my own personal experience, and could not represent the general opinion, because, according to Marchetti, 95 different surgical procedures have been devised and reported in the literature to correct stress incontinence, many in the last few years. Some of these procedures, unlike Dr. Kelly's, are difficult technically, and occasionally have been followed by sequelae such as fistulas and strictures which were worse than the original condition.

A new idea was introduced into this confused situation in 1949 when Kegel suggested the use of exercises for the relief of stress incontinence. He published a second paper on this subject in 1951. As far as we know, this was the first time that this form of treatment was used in this condition in women, although urologists tell me that they have been using it for many years to improve urinary control after prostatectomy and other operations on the male urethra. Likewise, rectal surgeons have been using these exercises after operations upon the rectal sphincters. Kegel reported remarkable success in his cases of stress incontinence, and this stimulated me to study the problem in my own private practice.

This report is based on the cases of stress incontinence that I have seen, mostly in the past three years. It also includes two ward cases treated by the resident staff of the Johns Hopkins Hospital and one treated by the residents of the

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Women's Hospital. This series includes 60 cases; this report, however, is based on the first 40.

In all of these women, stress incontinence was a major complaint. They were all subjected to a complete gynecological study and any other indicated investigations. It was my object to try to discover any factors that were associated with the stress incontinence, to try to correct them when possible and to observe the effect of this therapy on urinary control. It was my particular desire to try to discover whether non-operative measures, especially exercises, were helpful in this condition. We shall first summarize the clinical data discovered in these cases.

#### CLINICAL DATA

There is nothing new or startling in these clinical facts.

*Age.* Although stress incontinence may develop in early life, 75 per cent of our cases were between 40 and 60 years of age. Two were over ninety years old; two between twenty and thirty.

*Marital.* Thirty-seven of these women were married; three were single.

*Parity.* Thirty-three had borne children; seven had never been pregnant.

#### DURATION OF INCONTINENCE

0-1 year	6 cases
1-5 years	9 cases
5-10 years	4 cases
10-20 years	9 cases
20-30 years	4 cases
30-40 years	4 cases
More than 40 years	2 cases

#### ASSOCIATED DISEASES OF THE URINARY TRACT

In 10 patients, 25 per cent, there were the following urological disorders:

Urethral caruncle	1
Urethritis and trigonitis	4
Chronic cystitis	3
Chronic pyelitis and hydronephrosis	2

These conditions were all treated and were corrected, with the exception of one case of infected hydronephrosis with recurring acute pyelitis, which was only improved. In none of these

cases, however, was the stress incontinence completely cured by the elimination of the urological disorder. In all but two, however, the stress incontinence was definitely improved and control was better after the urological condition had been corrected. In two, a patient with urethral caruncle and another with chronic cystitis, the control was no better after than before the urological treatment.

From this, it is clear that the elimination of a urological disorder is helpful in correcting stress incontinence, on the basis that a normal urinary tract will function better than a diseased one. In all but two of these cases, even better control was obtained by the use of exercises after the urological treatment had been completed.

Strong, of Philadelphia, has reported better results than this, following the elimination of urologic disorders in women with stress incontinence—(In Discussion of paper in *Journal of Urology*, April, 1953.) There will be percentage differences in all such reports; the basic fact remains that in stress incontinence in women, it is advisable to make careful urologic studies when indicated, and to restore the urinary tract to normal before expecting much relief in urinary control.

#### GYNCOLOGIC DISORDERS FOUND IN CASES OF STRESS INCONTINENCE

In this series there were 14 cases showing various degrees of pelvic relaxation or prolapse. There were eight cases of complete prolapse of the uterus and vagina, and six with cystocele and rectocele.

In twelve of these cases of complete prolapse, the procidentia had been cured by appropriate surgical procedures. These operations had been performed by various gynecologists, chiefly in Baltimore. These patients continued to have stress incontinence, after having been cured of prolapse. We have frequently heard that in stress incontinence, associated with prolapse, one need only correct the prolapse, restore adequate sub-vesical support and the stress incontinence will then disappear spontaneously. This is undoubt-



edly true in many cases; in some, however, it is not the case, as this series shows.

In only four of these twelve, had any particular effort been directed toward correcting urinary control, and in these Kelly plication of the sphincter had been carried out in addition to the operation for prolapse. In three of these, the control had been improved; in one, there had been no improvement.

In these 12 women who still had stress incontinence after having been cured of uterine prolapse, we suggested exercises. Not all of them co-operated, but of the twelve, 5 were completely cured of stress incontinence and one was improved by exercises.

In this group with prolapse and stress incontinence, two were not operated upon. One was rather elderly, 74 years old, with complete procidentia, a ward patient in the Women's Hospital. While she was in the hospital, being studied preoperatively, she had a coronary thrombosis. This completely eliminated all further consideration of surgery. Since her main complaint had been stress incontinence, the resident staff gave her a course of exercises, which restored complete urinary control. In the second case, also, one of cystocele and rectocele, the main complaint had been stress incontinence. As a preoperative measure, exercises proved so successful that the patient has not been operated upon for the cystocele and rectocele.

#### POSTOPERATIVE STRESS INCONTINENCE

This series includes three cases of postoperative stress incontinence; two developed after vaginal hysterectomy for prolapse, and one developed after a Manchester operation. In none had it been present before the operation.

One of these cases is rather unusual in that she developed stress incontinence after each of two operations for prolapse and recovered complete control without any further treatment at all. The first time, stress incontinence appeared after a vaginal hysterectomy performed elsewhere; it lasted one year and then disappeared

spontaneously. Unfortunately, however, the vaginal hysterectomy was followed by vaginal prolapse, without any urinary symptoms. I operated upon her on the second occasion in 1949, and she again developed stress incontinence, which again disappeared spontaneously in one year. So far the prolapse has not occurred.

In the second case which developed after a vaginal hysterectomy, the patient did not try the exercises and still has stress incontinence. In the third case, which developed after a Manchester operation done by me, the stress incontinence disappeared completely after three weeks of exercises.

This group of cases of stress incontinence associated with prolapse or cystocele, leads one to suggest that stress incontinence is not always dependent on uterine prolapse, that a person may have complete prolapse without any stress incontinence, and that with or without prolapse, stress incontinence may occasionally be cured by strengthening the perineal and sphincter muscles by exercises. These data are susceptible of various interpretations.

#### STRESS INCONTINENCE AND VESICO-VAGINAL FISTULA

This association has been seen in one instance. In 1951, a 37 year old woman developed a vesico-vaginal fistula after an operation for a urethral diverticulum. This fistula persisted after the first operation to cure it; following this, she was sent to Baltimore and admitted to the Johns Hopkins Hospital as a ward patient. The resident gynecologist found not only a small vesico-vaginal fistula but also a weak vesical sphincter with stress incontinence. He closed the fistula and did a Kelly plication of the vesical sphincter. The fistula operation was a complete success, but the patient was almost as incontinent as ever, because of the failure of the sphincter operation. The resident therefore gave her urethral exercises, and since 1951, she has had excellent urinary control.



## GENERAL FACTORS

*Obesity.* Fifteen of these 40 incontinent women were more or less obese. In all of these women, we tried to interest them in diet control and weight reduction, usually with no success. In some instances, however, we have felt that part of the success of the treatment of stress incontinence was due to weight reduction.

*Neurological disorders.* An excellent operative result in stress incontinence may be completely vitiated by the development of a serious neurological disease. This happened to two of our patients, in whom excellent results had been obtained by Kelly plication followed by exercises. These patients later developed both rectal and urinary paralysis, in addition to mental changes as the result of serious central nervous system disease. Neurological examinations are always indicated in certain cases of stress incontinence, before starting any course of treatment.

*Psychic instability.* Experience teaches us that urinary control is affected by nervousness, even in normal women. When there is a weakness of the vesical sphincter or the control mechanism is somewhat impaired, nervous and psychic instability may also upset urinary continence. One of our patients has stress incontinence only while in Baltimore, in the midst of her domestic and business disturbances—while away from town she has perfect control. Other women state that excitement is just as effective as coughing or sneezing in producing incontinence. This is therefore a factor to be considered.

## THE TYPE OF EXERCISE USED

It is basically important that the patient understand what she has to do. In spite of all our explaining, we have found that some women have failed utterly to comprehend the situation.

The exercise is simply the same contraction that one makes when stopping the flow of urine. It is the same effort that one makes voluntarily to prevent the flow of urine, when the desire to void appears under unpropitious circumstances.

At the same time, one often contracts the rectal sphincter and perineal muscles.

This is the exercise the patient is to perform. She is told to contract the urethral sphincter in this way 15 times in the morning, 15 times at noon, and 15 times at night. She may also perform this exercise as often in addition as she wants to. She should do this for at least one month—the average case will show improvement before that.

*The perineometer.* I have not found the perineometer necessary. Kegel has used it almost routinely, but we have never used it. It would seem to us an unnecessary encumbrance that would prevent the use of exercises except at home. It would also greatly complicate the performance of simple exercises. Furthermore, our results without the perineometer have been about the same as the best results obtained with it. We see no reason, however, for withholding its use in any case where it would appear desired or advantageous.

## SUMMARY OF RESULTS

Following the various non-operative methods of treatment outlined previously in 33 cases, 11 have been cured, 13 improved and 9 unimproved.

Cured	11 cases	33%
Improved	13 cases	39%
Unimproved	9 cases	27%

Since in this situation, the result is based entirely on the functional result and is determined by the patient herself, the definition may not be as sharp as we would like. We have listed as cured only those who state that they have had no incontinence since using the exercises. The other terms are more or less self-explanatory. However, in our experience there is apt to be some lack of permanency in the definitions between these groups, in that a woman who may be perfectly continent for months may, under nervous or physical strain, have a recurrence of stress incontinence. We have seen this several

times; almost invariably these patients resume exercises for a few weeks with good results.

*Types of treatment used.* In this group we have used only non-operative measures. In 10 of the 33 patients, *urological disorders* were discovered and treated, with improvement in the stress incontinence in all but 2. After the urinary disorders had been corrected, the use of exercises gave even better or perfect urinary control. In other words, normal urinary control is more easily obtained with a normal urinary tract.

Other factors that have contributed to stress incontinence have been treated. This includes obesity, nervousness, gynecological, psychiatric and neurological disorders. It is our impression that the elimination of any associated factor will definitely aid in recovering urinary control and contribute to its permanence.

In practically all of these cases, exercises were used to complete the treatment and supplement the benefit that may have been obtained by other means.

On the basis of this and subsequent experience, we have the general impression that a perfect result obtained surgically by the Kelly plication operation or any other surgical means, is more apt to be permanent than an equally good result obtained by exercises. However, in both groups, an excellent result may be completely ruined by subsequent developments such as neurological diseases or an intractable cough due to asthma or chronic bronchitis.

#### CAUSES OF FAILURE

In analysing the cause of failure in 9 cases, we find the following: Senility, failure to perform the exercises, neurological disease, nervousness and psychic instability and coughing due to asthma and bronchitis.

Kegel pointed out the fact that senility and chronicity were unfavorable factors; that applies whether the stress incontinence is treated by surgical or non-operative measures. One of our failures following non-operative care, was in a spinster over 90 years old; another in a woman

over 75. It is difficult to get a woman 90 years old to perform exercises or alter a situation which has existed for decades, regardless of how inconvenient it may be.

Five women did not attempt the exercises at all. Some were fairly young and were apparently too lazy to do anything for themselves. In others, failure to cooperate was understandable because of their senility.

In two women, results that had been satisfactory were completely ruined by the development of organic paralysis of the rectal and urinary sphincters. Both of these women had had Kelly plications many years previously with subsequent recurrence of stress incontinence, which had been relieved by exercises.

The development of unusual stress or strain may likewise overcome a good result, either surgical or non-operative. In two of our cases that had been considered cured of stress incontinence by exercises, the development of asthma and chronic bronchitis with uncontrollable coughing, produced a recurrence of stress incontinence, which we have so far been unable to correct.

#### THE PLACE OF EXERCISES AND NON-OPERATIVE THERAPY IN THE TREATMENT OF STRESS INCONTINENCE

In reviewing our experience in this study of stress incontinence, we have gained the impression that stress incontinence may occur under almost any circumstances—in the parous and nulliparous, in women with prolapse and without any evidence of pelvic relaxation, in the young and the aged. It may persist after an otherwise successful correction of uterine and vaginal prolapse. It may appear postoperatively after the surgical correction of prolapse.

In other words, stress incontinence has been found to occur under the most diverse circumstances. Whether there is any common denominator among these factors, we have not been able to determine, unless perhaps it be a weakness of the sphincter mechanism. This has appeared to us to be the most likely common factor, inas-

much as under all these diverse conditions, exercises have in the majority of instances improved urinary control. Whether these exercises might relieve stress incontinence in any other way, we cannot tell.

In view of this experience, and also the experience of Kegel, it appears that stress incontinence may be more than a surgical problem. We have listed some of the factors that seem to have played a part in its production and our results in eliminating these factors before operative treatment is considered. In general, we believe that in any person who complains of stress incontinence and in whom there is no other indication for surgical treatment, non-operative treatment should be tried first, as we have suggested. In our experience, in three-fourths of these cases, the benefit of non-operative care will be so great that an operation will not be needed. If, however, in a patient with stress incontinence, a condition is found which might contribute to poor urinary control or in itself require surgical correction, such as uterine or vaginal prolapse, under such conditions we would perform the indicated surgical procedure and at the same time do a Kelly plication of the vesical sphincter or one of the other satisfactory procedures of this sort. If, after this surgical treatment, urinary control is still poor, we would attempt to determine its cause and institute proper treatment. In a number of such cases, exercises have been very helpful. In other words, in our experience, such non-operative measures have proved themselves a useful addition in our

methods of treating stress incontinence in women.

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## Component Medical Societies

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### ALLEGANY-GARRETT COUNTY MEDICAL SOCIETY

LESLIE E. DAUGHERTY, M.D.

*Journal Representative*

Dr. A. Wolferman, Frostburg physician, is now serving a three year residency at the Eye, Ear, Nose and Throat Hospital in Baltimore. Dr. Wolferman attended medical school in Switzerland and graduated from the University of Geneva in 1940. Following graduation he served one year of internship and two years of residency in internal medicine at the University Hospital, of the City of Geneva. He was Assistant Superintendent of a Tuberculosis Hospital in Switzerland for two years and a resident physician at the Maryland Tuberculosis Sanatorium for two years. From 1948 until July, 1953, Dr. Wolferman did general practice in Frostburg, belonging to the staff of the Miners Hospital in Frostburg and the Sacred Heart Hospital, in Cumberland.

Dr. Leo H. Ley, of Cumberland has been appointed City Physician, to replace Dr. John K. Rozum who resigned two months ago shortly before moving to Florida. Dr. Ley has been practicing in Cumberland about 15 months. He is a veteran of World War II and completed medical studies at the University of Maryland following service with the armed forces. Dr. Ley served his internship and residency at St. Agnes Hospital, Baltimore, Md.

Dr. E. I. Baumgartner, of Oakland, Md. spoke at a recent meeting of the Woman's Civic Club, Oakland. The title of his subject was "The Golden Era of Therapeutics." Dr. Baumgartner is secretary of the Section on General Practice of the American Medical Association and a member of the board of directors of the American Academy of General Practice. He is also a member of the Town Council of Oakland and Deputy Medical Examiner.

The following article appeared in the Cumberland Evening Times, Aug. 9, 1953:

#### DR. FRANTZ EXPLAINS FEAR OF POLIO OFTEN EXCESSIVE

Don't be too worried about poliomyelitis, Dr. Winter R. Frantz, county health officer, advised

today. Polio is not as common and usually not as dangerous as many other less publicized illnesses, he explained adding that rheumatic fever, tuberculosis, and many handicapping conditions, such as accidental injuries, tumors, epilepsy, cerebral palsy and a host of others are more to be feared. No cases have been reported in Allegany County this year.

During the last four polio epidemics in Maryland, in 1944, 1949, 1950 and 1952, Dr. Frantz stated the total number of reported cases showing any sign of paralysis averaged only 341 for the entire state during the epidemic year, or only one case in about 2,300 of the population under age 20. Moreover, he continued about 76 per cent of the cases diagnosed as paralytic poliomyelitis eventually recovered with little or no handicap. On the basis of these statistics, Dr. Frantz said the risk of contracting severely paralyzing polio is only about 1 in 10,000, even during an epidemic year.

Deaths and severe disabilities resulting from rheumatic fever, accidents and tuberculosis far exceed those caused by polio, Dr. Frantz pointed out.

Knowledge of the facts of polio often helps to calm fears, he said. "We know," he continued, "that transmission of the disease for the great majority of cases is by association with infected persons and that there are approximately 200 non-paralytic infections to every paralytic case. During any outbreak, therefore, the virus is so widely spread that quarantine methods other than prompt isolation of recognized cases is useless," Dr. Frantz commented.

The health officer pointed out current scientific evidence compiled at Johns Hopkins University indicates ultimate escape from infection is impossible and that temporary escape is undesirable as it postpones infection to later ages when its consequences are more severe.

Dr. Frantz also explained that people 15 years of age and older usually build up antibodies which protect them from at least one of the known polio viruses. During childhood, he said, contacts usually unknown at the time give this immunity to polio viruses. Children who have no reserve of antibodies, Dr. Frantz continued, are more susceptible than adults for this reason.

Gamma globulin, the blood derivative being used as a temporary polio preventive, will be available in Maryland only to children under 18 years and to pregnant women living in the same individual household with a proven polio victim, except in the case of a recognized epidemic, Dr. Frantz said. If advisable, he added, mass inoculations will be given in the high-risk age groups where there is an epidemic.

Gamma globulin, which induces immunity for approximately five weeks, is at best temporary preventive, Dr. Frantz pointed out, explaining that there is no cure for polio once the nerve cells have been affected. The prospects are bright for active immunity, he said, in view of recent scientific progress.

"Above all don't become hysterical about polio," he cautioned.

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### Baltimore City Medical Society and its Sections

Meetings will be held at 1211 Cathedral Street unless otherwise stated

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### BALTIMORE CITY MEDICAL SOCIETY

*Friday, November 20, 1953, 8:30 p.m.*

#### CO-SPONSORSHIP WITH THE HEART ASSOCIATION OF MARYLAND

*8:45 p.m.*

The Medical Management of Acute and Chronic Anterior Occlusion. A. Wilbur Duryee, M.D., Professor of Clinical Medicine, Postgraduate Medical School of the New York University-Bellevue Medical Center, New York City.

*9:30 p.m.*

The Physical Aspects of Peripheral Vascular Disease. Jere W. Lord, Jr., M.D., Associate Professor of Clinical Surgery, Postgraduate Medical School of the New York University-Bellevue Medical Center, New York City.

*10:15 p.m.*

Question period.

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### SECTION ON DISEASES OF THE CHEST

A. MURRAY FISHER, M.D., *Chairman*

EDMUND G. BEACHAM, M.D., *Secretary*

*Wednesday, November 4, 1953, 8:00 p.m.*

Emotional Aspects of Pulmonary Tuberculosis. Jerome Hartz, M.D., Assistant Professor of Psychiatry, The Johns Hopkins University School of Medicine.

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### OTOLARYNGOLOGICAL SECTION

C. CARLETON DOUGLASS, M.D., *Chairman*

ALBERT STEINER, M.D., *Secretary*

*Tuesday, November 10, 1953*

*Johns Hopkins Club, Homewood Campus*

*Cocktails 6:00 p.m. Dinner 6:30 p.m.*

Papers will be presented by the Residents of the various hospitals.



**MATERNAL MORTALITY COMMITTEE**

*Thursday, November 19, 1953, 4:00 p.m., Osler Hall*

Joint Committee on Maternal Mortality of the Baltimore City Medical Society and the Baltimore City Health Department.

**THE COMMITTEE FOR THE STUDY OF PELVIC CANCER**

Sponsored by the Maryland Division of the American Cancer Society and the Medical and Chirurgical Faculty.

RICHARD W. TELINDE, M.D., *Chairman*

BEVERLEY C. COMPTON, M.D., *Secretary*

*Thursday, November 19, 1953, 5:00 to 6:00 p.m.*

**DERMATOLOGICAL SECTION**

EUGENE S. BERESTON, M.D., *Chairman*

RAYMOND C. V. ROBINSON, M.D., *Secretary*

*Monday, November 23, 1953, 8:30 p.m.*

1. Election of Officers.
2. Experimental Contact Dermatitis with Dinitrochlorobenzene. (Illustrated.)  
Israel Zeligman, M.D.

**DORCHESTER COUNTY  
MEDICAL SOCIETY**

WALTER B. JOHNSON, M.D.

*Journal Representative*

The Dorchester County Medical Society usually meets the third Wednesday of each month, but discontinues meeting for June, July, and August.

On July 22nd, however, Dr. John Mace, Jr., of Cambridge, entertained the society at his summer home at Greenpoint on the Choptank River, to a crab feast. There was no business meeting, but a very enjoyable social evening was held.

•Dr. George Currier, who was appointed Superintendent of the Eastern Shore State Hospital, and who formerly was a member of the Massachusetts State Medical Society has applied for membership in the local county and state associations.

Dr. Thomas Dredge, Clinical Director at the Eastern Shore State Hospital, also presented a card of transfer from the Green Bay, Wisconsin Medical Society.

Dr. Wilbur Baumann who was recently released from the United States Navy has applied for membership in the county and state societies.

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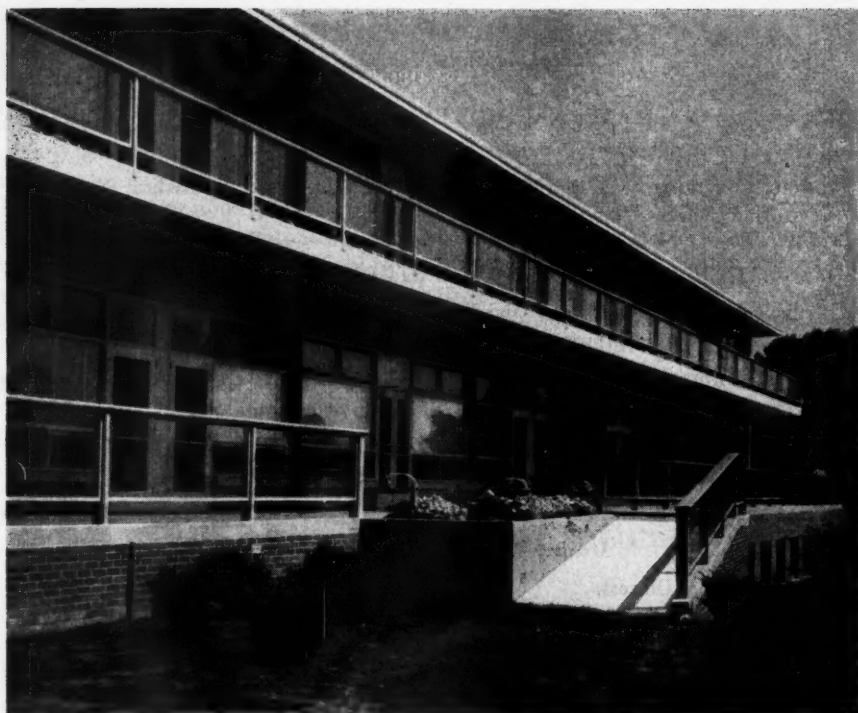
**REQUESTING THAT ALL DEATHS ASSOCIATED WITH PREGNANCY BE REPORTED!**

JOHN WHITRIDGE, JR., M.D.\*

The Committee on Maternal and Child Welfare of the Medical and Chirurgical Faculty has for many years been reviewing all maternal deaths among residents of the counties of Maryland. Recent changes in statistical classification of deaths have raised certain difficulties in regard to whether a death should be officially classified as maternal or non-maternal. The Committee is anxious to review *all* adult deaths associated with pregnancy. Such deaths, whether classified as maternal, or non-maternal, would be tabulated annually as "Total Deaths Associated with Pregnancy."

In order to make this comprehensive review possible, all physicians are accordingly urged to indicate in an appropriate place on death certificates the fact that patients were or had recently been pregnant, even though, in the reporting physician's opinion, pregnancy or delivery may have played no role whatever in causing death.

\* Secretary, Committee on Maternal and Child Welfare, Medical and Chirurgical Faculty.



### NEW MOUNT PLEASANT HOSPITAL NOW OPERATING IN BALTIMORE\*

Baltimore's new Mount Pleasant Sanitorium, a fifty-four bed hospital, located at Greenspring and Belvedere Avenues, is an example of a completely self-contained specialized tuberculosis hospital integrated into a modern medical center. The national magazine, "Modern Hospital," has characterized Mount Pleasant as an outstanding example of a modern up-to-date hospital for tuberculosis and other pulmonary diseases.

Mount Pleasant is the third completed unit of the \$16,000,000 Jewish Medical Center expansion program of the Associated Jewish Charities. Also, already completed are two new buildings at Levindale, a home for the aged, a new dining hall and a new fifty bed infirmary addition, as well as the Mount Pleasant Building. The entire Jewish Medical Center is being built on grounds adjacent to Levindale. Final plans for the new Sinai, now located at East Monument Street, are expected to be ready by the Fall of 1954, at which time it is expected that ground will be broken for Sinai's first six buildings.

\* Henry W. Levy, Publicity Director, Associated Jewish Charities.

The new Jewish Medical Center, comprising 150 acres that includes four corners at Belvedere and Greenspring Avenues, will give Baltimore its third major medical center. It will have a total capacity in the three autonomous hospitals of 745 beds, 231 at Levindale, 54 at Mount Pleasant and 460 at Sinai.

Built at a cost of \$1,400,000, the fifty-four bed Mount Pleasant is divided into eighteen double rooms and sixteen single rooms, almost all of which open on to porches and terraces, and all of which have private toilet facilities. The hospital also contains its own dining-room, kitchen, auditorium, Synagogue, library, radiology department, oxygen room, motion picture projection room and occupational therapy facilities.

Mount Pleasant, which was founded by the late Jacob Epstein in 1908, was formerly located on a 135-acre tract of land in Reisterstown, Md., twenty miles outside of Baltimore. The property, which is now being offered for sale or rent by the Associated Jewish Charities, includes a hospital building, dining hall, nurses' dormitory and several brick cottages as well as farm buildings.

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## Library

"Books shall be thy companions; bookcases and shelves, thy pleasure-nooks and gardens." *ibn Tibbon*

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### EPILEPSY

LOUIS KRAUSE, M.D.\*

This month's publication of books will be devoted to the treatises on Epilepsy. This is a very ancient disease, having been called the "sacred disease" by the ancients; *sacra medica* by the Romans or *Morbis Comitialis*. In English, this is called comitial or congress disease. This is another instance of disease, the description of which we have very graphically presented in our Bibles, particularly in the New Testament. In Mark, 9th chapter, 17th verse, we have the following quotation, "... Master, I have brought unto thee my son, which hath a dumb spirit;

Verse 18 "And wheresoever he taketh him, he teareth him: and he foameth, and gnasheth with his teeth, and pineth away. ...

Verse 20 "... and when he saw him, straightway the spirit tare him; and he fell on the ground, and wallowed foaming."

Verse 21 "And he asked his father, How long is it ago since this came unto him? And he said, Of a child."

Verse 22 "And oftentimes it hath cast him into the fire, and into the waters, to destroy him. ...

Verse 25 "... Thou dumb and deaf spirit, I charge thee, come out of him, and enter no more into him.

Verse 26 "And the spirit cried, and rent him sore, and came out of him.

Again another reference of the same incident is in the Book of Luke, Chapter 9, Verse 39 "And, lo, a spirit taketh him, and he suddenly crieth out; and it teareth him that he foameth again, and bruising him hardly departeth from him."

These passages describe the essential manifestations of the disease as we know it even today, the only thing lacking being the electroencephalogram.

An amazing description of the sacred disease was given by Hippocrates which really was the highest reach of free thought for centuries; and had his statements been heeded, it would have done away

with the foolish notion that human ills were caused by gods and demons and certainly with the notion that epilepsy was of divine origin.

The disease has often been referred to the phases of the moon and some mental diseases were classified as diseases related to the changes of the moon. Hence the word *lunatic*. Likewise, this is one of the reasons why silver nitrate was used at one time in the treatment of epilepsy because of silver being the moon metal and it was thought to be efficacious in such diseases.

The present term, *Epilepsy*, comes from the Greek meaning *seizure upon*. The following list of books which are available in our Library will afford much interesting study and reading on this disease, which is more effectively treated today than ever before:

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\* Chairman, Library Committee.

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## LIBRARY NOTES

We report with regret the resignation, effective September 30th, of Mrs. Eleanor Kohler, who has been a most valuable assistant in the Library since May 1949. Her quick and intelligent help, her enthusiasm, and her cheerful disposition will be missed by everyone, and she carries with her all good wishes for her happiness in the family life she has chosen for her career.

We welcome her successor, Miss Myrtle Hollins, and wish her happiness and success in her work in the Library.

## A SELECTED LIST OF PUBLICATIONS RECENTLY ADDED TO THE LIBRARY

- Armstrong, J. R.** Lumbar disc lesions: pathogenesis and treatment of low back pain and sciatica. Baltimore, Williams and Wilkins Co., 1952.
- \***Baer, K. A.** Pathology and physiology of burns: an annotated bibliography, 1942-1951. Washington, Armed Forces Medical Library, 1952.
- Baldwin, E.** Dynamic aspects of biochemistry. 2d ed. Cambridge, University Press, 1952.
- Brock, S.** Basis of clinical neurology. 3d ed. Baltimore, Williams and Wilkins Co., 1953.
- \***Browning, E.** Toxicity of industrial organic solvents. London, H. M. Stationery Office, 1953.
- Cantor, A. J.** Ambulatory proctology. 2d ed. New York, Hoeber, 1952.
- Cattell, R. B. and Warren, K.** Surgery of the pancreas. Philadelphia, W. B. Saunders, 1953.
- \***Cavina, C.** Indicazioni ed i metodi della resezione epatica. Bologna, Cappelli, 1952.
- Clark, G. W.** Vitamin digest. Springfield, C. C. Thomas, 1953.
- Coope, R.** The quiet art, a doctor's anthology. Edinburgh and London, Livingstone (Baltimore, Williams & Wilkins), 1952.

\* Indicates gifts.



- Courville, C. B. *Commotio cerebri*. Los Angeles, San Lucas Press, 1953.
- Dale, P. M. *Medical biographies: the ailments of 33 famous persons*. Norman, University of Oklahoma Press, 1952.
- Decker, A. *Culdoscopy, a new technique in gynecologic and obstetric diagnosis*. Philadelphia, W. B. Saunders, 1952.
- Gross, R. E. *Surgery of infancy and childhood*. Philadelphia, W. B. Saunders, 1953.
- Johnson, J. and Kirby, C. K. *Surgery of the chest*. Chicago, Year Book Publishers, 1952.
- Jukes, T. H. *B-vitamins for blood formation*. Springfield, C. C Thomas, 1952.
- Lewin, P. *The knee and related structures*. Philadelphia, Lea and Febiger, 1952.
- Maingot, R. *Management of abdominal operations*. New York, Macmillan Co., 1953.
- \*Merck and Company. *Annotated bibliography on cortone*. Rahway, Merck and Co., 1953.
- \*Moloney, J. C. *The battle for mental health*. New York, Philosophical Library, 1952.
- Monro, T. K. *The physician as man of letters, science and action*. 2d ed. Baltimore, Williams and Wilkins Co., 1951.
- Najjar, V. A. *Carbohydrate metabolism*. Baltimore, Johns Hopkins Press, 1952.
- Selye, H. *Physiology and pathology of exposure to stress*. Montreal, Acta, Inc., 1950.
- Sharpe, W. *Brain surgeon: the autobiography of Dr. William Sharpe*. New York, Viking Press, 1952.
- Simpson, K. *Modern trends in forensic medicine*. St. Louis, C. V. Mosby, 1953.
- Slocum, D. B. *Atlas of amputations*. St. Louis, C. V. Mosby, 1949.
- Thomas, L. *Rheumatic fever*. Minneapolis, University of Minnesota Press, 1952.
- Thorek, P. *Anatomy in surgery*. Philadelphia, J. B. Lippincott, 1951.
- Todd, J. C., Sanford, A. H. and Wells, B. B. *Clinical diagnosis by laboratory methods*. Philadelphia, W. B. Saunders, 1953.
- Welch, C. E. *Surgery of the stomach and duodenum*. Chicago, Year Book Publishers, 1951.
- Williams, H. L. *Menière's disease*. Springfield, C. C Thomas, 1953.
- Windle, W. F. *Asphyxia neonatorum*. Springfield, C. C Thomas, 1950.
- Wolf, S. and Wolff, H. G. *Headaches, their nature and treatment*. New York, Little, Brown and Co., 1953.
- Zeigler, R. F. *Electrocardiographic studies in normal infants and children*. Springfield, C. C Thomas, 1951.

\* Indicates gifts.

# STUDY OF VA MEDICAL CARE MAY GO OVER TO NEXT SESSION

AMA Washington Letter, No. 28

The House Veterans Affairs subcommittee study of the knotty problem of VA medical care for non-service-connected cases may take much longer than originally anticipated. First indication of this came on the opening day of hearings when Chairman Pat Kearney (R-N.Y.) commented that he wanted all veterans' groups to make concrete recommendations on the issue "after their conventions this fall." Rep. Kearney cautioned that unless the committee came up with some specific legislation, Congress itself "might jam something through that we will all be sorry for." Among questions the committee is seeking answers to are: What is the definition of service-connected disability? How far should Congress go beyond the present GI Bill of Rights in saying what the government owes in non-service-connected cases? Would enforcement of the existing inability-to-pay provisions for non-service cases be preferable to new legislation?

Testimony of veterans' groups up to this point indicated that there is no unanimity of opinion on the problem. The Veterans of Foreign Wars promised some stand would be taken at the annual encampment in August; the Disabled American Veterans felt that better policing of existing regulations is preferable to new legislation; the AMVETS advocated clear-cut language in a law to wipe out abuses.



STATE OF MARYLAND DEPARTMENT OF HEALTH  
MONTHLY COMMUNICABLE DISEASE REPORT

Case Reports Received during 4-week Period, August 28-September 24, 1953

	CHICKENPOX	DIPHTHERIA	GERMAN MEASLES	HEPATITIS, INFECT.	MEASLES	MENINGITIS, MENINGOCOCCUS	MUMPS	POLIOMYELITIS, PARA- LYTIC	POLIOMYELITIS, NON PARALYTIC	ROCKY MT. SPOTTED FEVER	STREP. SORE THROAT INCL. SCARLET FEVER	TYPHOID FEVER	UNDULANT FEVER	WHOOPING COUGH	TUBERCULOSIS, RESPIRATORY	SYPHILIS, PRIMARY AND SECONDARY	GONORRHEA	OTHER DISEASES	DEATHS Influenza and pneumonia
Total, 4 weeks																			
Local areas																			
Baltimore County.....	2	—	—	17	—	1	9	18	18	—	1	—	—	5	29	1	12	a-1	7
Anne Arundel.....	2	—	—	1	—	—	1	6	3	—	—	—	—	—	9	—	4	—	1
Howard.....	—	—	—	—	—	—	—	4	1	—	—	—	—	—	2	—	4	—	—
Harford.....	1	—	1	2	2	—	2	4	1	1	—	1	—	8	2	—	1	—	1
Carroll.....	—	—	—	2	2	—	1	1	—	1	—	—	—	—	—	—	—	—	6
Frederick.....	—	—	2	1	—	—	—	3	3	—	2	—	—	3	—	—	—	—	—
Washington.....	1	—	—	—	2	—	—	—	—	—	—	—	—	1	7	—	2	—	—
Allegany.....	—	—	—	3	4	—	1	—	—	2	—	2	—	5	1	—	—	—	1
Garrett.....	—	—	—	2	—	—	—	2	—	—	—	—	—	—	1	—	—	—	—
Montgomery.....	—	—	2	—	—	—	2	6	6	1	1	—	—	—	9	—	—	—	2
Prince George's.....	—	—	1	5	—	—	4	5	5	—	—	—	—	2	6	—	3	m-1	3
Calvert.....	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—
Charles.....	—	—	—	—	—	—	—	1	2	—	—	—	—	—	2	—	2	—	—
Saint Mary's.....	—	—	—	16	—	—	—	—	1	—	—	—	—	—	—	—	2	—	—
Cecil.....	—	—	—	—	—	—	—	1	—	—	—	—	—	—	3	—	1	m-1	1
Kent.....	2	—	—	1	—	—	—	—	—	—	—	—	—	—	—	—	1	—	—
Queen Anne's.....	—	1	—	—	—	—	—	1	2	—	—	—	—	—	—	—	—	—	—
Caroline.....	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	1	—	—
Talbot.....	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	1	—	1
Dorchester.....	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	12	—	—
Wicomico.....	—	—	—	1	3	—	—	—	—	—	2	—	—	1	1	—	25	—	2
Worcester.....	—	—	—	—	—	—	—	—	2	—	—	—	—	2	—	2	1	c-1	—
Somerset.....	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1	—	5	—	—
Total Counties.....	8	1	6	51	13	1	20	50	47	5	6	3	0	28	74	4	77	—	25
Baltimore City.....	7	0	5	4	17	1	34	29	25	0	6	0	0	49	102	8	668	—	18
State																			
Aug. 28-Sept. 24, '53...	15	1	11	55	30	2	54	79	72	5	12	3	0	77	176	12	745	—	43
Same period 1952.....	7	0	8	17	10	5	37	49	34	3	17	0	0	9	194	25	680	—	23
5-year median.....	15	2	5	—	29	1	23	55	—	7	20	4	2	78	197	53	705	—	28
Cumulative totals																			
State																			
Year 1953 to date.....	2698	10	1426	384	1488	60	2235	218	175	23	2181	22	9	300	1772	106	5864	—	567
Same period 1952.....	2736	7	831	174	9068	71	910	92	70	28	836	15	12	160	2020	146	5374	—	498
5-year median.....	3039	57	525	—	3983	59	1586	—	145	53	878	25	33	607	2066	652	5515	—	470

a = amoebic dysentery.

c = congenital syphilis under 1 year of age.

m = malaria contracted outside of Maryland.



# Blue Cross - Blue Shield



## BLUE CROSS AND BLUE SHIELD CONTINUE TO GROW

R. H. DABNEY\*

During the first six months of 1953, Blue Cross payments for hospital care totalled \$5,821,103, an all-time high for the fifteen years the Plan has been in operation.

These payments were made for hospital services provided to 60,193 subscribers of the Plan. During the same period a year ago, 56,426 subscribers received care for which Blue Cross paid \$5,157,891. Hospital costs have gone up and more people are going to the hospital.

The average length of stay for patients was 7.3 days, and payments on individual cases ranged from \$25 to over \$2,000 for long-stay serious illnesses.

During the first half of the year, payments for hospital care amounted to 92.7 cents out of every dollar received, and only 6.4 cents out of each dollar was used for operating expenses. Membership in Maryland's Blue Cross Plan as of June 30 was 869,000, an all-time high and an increase of 10,000 subscribers since the first of the year. Nationally, there are now over 44 million subscribers.

Meanwhile, membership in Blue Shield continued to grow. There are now 200,000 subscribers, an increase of 12,000 since January 1.

Publication of these figures made the position of voluntary health plans in the national health picture subject of an editorial in the August 5 Balti-

more Sun. "A popular argument for national compulsory health insurance has been that no voluntary plan can be made to cover a sufficiently large proportion of the population. In conflict with this theory is the recent phenomenal growth of volunteer plans and, in particular, Blue Cross and Blue Shield," the editorial begins.

The editorial points out that at present nearly 36 per cent of Maryland's population is covered by Blue Cross compared to national membership of 27.34 per cent of the population. In Rhode Island Blue Cross membership takes in 78 per cent of the state's population. Membership continues to grow, in spite of the fact that enrollment must be by groups and, in consequence, there are many individuals who are ineligible. All this would seem to refute the argument that no voluntary plan can be made to cover a sufficiently large proportion of the population.

The editorial concludes, "Blue Cross and Blue Shield are, of course, only a part of the prepayment picture. According to the report of the Truman Health Commission, commercial insurance company payments directly related to medical care expenses and specifically paid for the purpose of defraying those expenses amounted in the single year of 1951 to \$768,000,000. When the American public shows so impressive an inclination to insure itself against the costs of medical care, surely it is premature to press for compulsory insurance as the only solution of the problem."

\* Executive Director, Maryland Hospital Service, Inc., Maryland Medical Service, Inc.

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# Woman's Auxiliary to the Medical and Chirurgical Faculty

MRS. CHARLES H. WILLIAMS, *Auxiliary Editor*

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## PRESIDENT'S MESSAGE

MRS. JOHN G. BALL

I pledge my loyalty and devotion to the Woman's Auxiliary to the American Medical Association. I will support its activities, protect its reputation and ever sustain its high ideals.

This is the pledge we say at Auxiliary meetings and which is always most impressive at the national conventions. There, we hear voices from every state in the union, Hawaii and Alaska pledging themselves to the work of the Auxiliary.

Now, just what is that work? Those high ideals? Our Constitution states our objects to be: (1) Through our members to extend the aims of the medical profession to all organizations which look to the advancement of health and health education; (2) To fulfill such functions as may be desired from time to time by the Medical and Chirurgical Faculty of the State of Maryland; (3) To promote acquaintanceship among physicians' families that fellowship may increase and at all times to stimulate a feeling of local cooperation. Each of these objects is important and vital to the medical profession in Maryland. No one member or one county can do this alone. Accomplishment of our objects depends on the willingness of each individual member to do her part.

At the National Convention in New York, June 1-5, 1953, we were shocked to learn that Maryland is at the very bottom of the list for Woman's Auxiliary Membership Percentage of the American Medical Association membership, that percentage being 23.5%. Now is the time for us, each and every one of us, to get busy and find those friends of ours in unorganized counties, visit them, talk with them about the Auxiliary, tell them our objects, the work we have been doing, and how rewarding the contacts and friendships we make, become. Your Organization Chairman, Mrs. Albert Goldstein, and your

President will be happy to visit or have any suggestions you may have. The most successful membership and organization work has just one secret—"personal contact and interest." At the Presidents' Conference following the New York meeting, Mrs. Leo J. Schaefer, our National President, gave us this theme: "Together we progress." Let us work together for progress in membership and organization.

As we strive for more members and more organized counties, let us not forget the work we have already started that needs our continuing support. In New York, we were proud to report our successful Future Nurses Convention held in Baltimore on May 12, 1953, when nearly 400 enthusiastic high school girls heard speakers on all phases of nursing, saw American Medical Association nursing films, and heard student nurses, in uniform, from 14 hospitals speak. All our organized counties have nursing scholarships. A Nurse Recruitment film is being made of the nursing schools in Maryland and will be ready for use this winter. Some of the high schools have Future Nurse Clubs which interest and teach the girls about nursing. More county Auxiliaries should sponsor these groups. In these ways we are meeting the challenge of the shortage of nurses which the American Medical Association has requested of us.

There are other continuing challenges for us; one of the greatest is in the field of Public Relations or "Public Responsibility" as one state has so aptly paraphrased it. Each doctor's wife needs to be informed and keep informed so she may be prepared to talk with the lay persons she meets daily, when questions are asked. Have you noticed that you can scarcely pick up a magazine but what there is some challenging article related to medicine, physicians, medical fees, malpractice suits, doctor shortage, emergency service and a host of other questions vital to us as physicians' wives and Auxiliary members. As individuals, we participate in many activities of

educational, civic and religious organizations. All of these contacts are most important and should be extended. Public service is the ultimate goal of public relations. How best may we serve our communities in the field of health? The American Medical Association has been increasing its public service by developing voluntary health insurance, doctor placement service, emergency call systems, exposing quacks and continuing to look for a solution to the problem of the chronically ill, aging, and mentally ill. Above all, we must continue to maintain the prestige and esteem of the medical profession in the eyes of the public.

Civil Defense needs our continuing greater interest and effort. Much of the organizational work has been completed so we should be able to see definite progress this year. Each one should find the place she may best help in her local organization.

Last year the American Medical Association asked the Auxiliary to help the American Medical Education Foundation with the problem of raising money for the medical schools of the Country, most of which have large deficits. The National Auxiliary gave \$10,000 to the Foundation at the New York meeting. Efforts must be continued to meet the goal of \$2,000,000 for this year. Let us continue to urge all physicians to subscribe to their medical schools through the American Medical Education Foundation. All such money given by physicians through the Foundation is matched by funds subscribed by private industry. Speak to your husband about this.

To help us in all these important tasks, we have the "Bulletin of the Woman's Auxiliary to the American Medical Association." Each National Chairman through her contacts throughout the States and through the States to the Counties has a wealth of information and help to offer each of us. The only way we can avail ourselves of this help is to subscribe to the "Bulletin." Until we all start reading and using the material presented to us, we are not beginning to do our job. If you do not subscribe, please do. The Bulletin Chairman, Mrs. S. Jack Sugar, 6709 41st Avenue, University Park, Maryland, will be happy to take your subscription for \$1.00.

Another object of the Auxiliary was fulfilled with the Semiannual Meeting. The Auxiliary helps with the planning and entertainment of all such meetings, as requested by the Medical and Chirurgical Faculty.

## PRESIDENTIAL APPOINTMENTS

Mrs. John G. Ball, President, has appointed the following Chairmen:

Standing Committees: "Today's Health," Mrs. S. R. Wells, Hagerstown; Legislation, Mrs. H. Hanford Hopkins, Ruxton; Organization, Mrs. Albert E. Goldstein, Baltimore; Public Relations, Mrs. George H. Yeager, Baltimore; Program, Mrs. T. A. Christensen, College Park; Finance, Mrs. Elliott C. Flick, Towson; Historian, Mrs. P. S. Lansdale, Frederick; Revisions and Resolutions, Mrs. Amos R. Koontz, Garrison; Doctor's Day, Mrs. Gerald Le Van, Boonsboro; Press and Publicity, Mrs. Charles H. Williams, Pikesville; Bulletin, Mrs. S. Jack Sugar, University Park; Members-at-Large, Mrs. James T. Marsh, Westminster.

Special Committees: Civil Defense, Mrs. Arthur Baptisti, Jr., Hagerstown; Nurse Recruitment, Mrs. James Kerr, Damascus; Medical Education Foundation, Mrs. J. Carlton Wich, Baltimore; Membership, Mrs. Martin Strobel, Reisterstown; Convention Arrangements, Mrs. George E. Urban, Catonsville; Auxiliary Editor (Maryland State Medical Journal) Mrs. Charles H. Williams, Pikesville.

## BALTIMORE COUNTY AUXILIARY AWARDS THIRD NURSING SCHOLARSHIP

The Woman's Auxiliary to the Baltimore County Medical Society awarded their third Nurse's Scholarship to Miss Mary Hughes of Catonsville. Dr. A. B. Daugharthy (the family physician of Miss Hughes) presented the award which will enable this student to attend the Johns Hopkins School of Nursing in September.

## MARYLAND AUXILIARY GIVEN NATIONAL RECOGNITION

### MARYLAND AUXILIARY PLANNING NURSE RECRUITMENT FILM

Reprinted from A.M.A. Woman's Auxiliary Bulletin Dec. 1952

As a public relations project, the Maryland Auxiliary, in cooperation with the local hospitals schools of nursing, state medical association and the nursing association, is making its own nurse recruitment movie. The idea originated in the Auxiliary and has

been enthusiastically received. Financing and script writing are now in the hands of committees composed of members of the Auxiliary, the medical association, representatives from hospitals and nursing associations, and civic organizations. In the tentative plan presented to the cooperating groups, the Auxiliary stressed the point that the film would portray nursing *not as a selfish interest but as a service profession emphasizing ethics as is done in the practice of medicine*. The Auxiliary is looking forward to a friendly

association with the hospital and nursing associations working together for the best interests of the community.

**STUDENTS ADMITTED TO ALL  
STATE-APPROVED SCHOOLS OF  
NURSING DURING 1950, 1951  
AND 1952**

Maryland—1950, 729; 1951, 828; 1952, 765.

**AMERICAN COLLEGE OF SURGEONS**

The Board of Regents of the American College of Surgeons has established the first of several contemplated scholarships in the field of research for promising young men seeking a career in academic surgery, and who have recently finished or who are in the final months of their residency training program.

Because surgeons have indicated their growing interest in research, and because of the vital importance of surgical research, it is hoped that this program will encourage and stimulate the interest of young surgeons with investigative ability and promise, and direct their development toward the pursuit of academic surgery.

Although the College is able to support only one scholarship at the present time, it is believed that through the efforts of Fellows and various organizations interested in this project support of additional research scholarships will be assumed.

Candidates must obtain the approval of the chairman of the department of surgery, dean of the same medical school, and the authority of an executive officer of the university making the proposal. Preference will be given to an American or Canadian citizen.

Successful candidates will receive \$20,000 over a three-year period, to be divided as follows: \$6,000 for the first year, \$6,500 for the second year, and \$7,500 for the third year. The medical school or institution sponsoring a successful candidate may supplement this amount with the permission of the Committee on Selection of the American College of Surgeons.

Medical schools or institutions supporting the nomination of a candidate will be required to provide a satisfactory place to work, with adequate facilities, as well as funds with which to support necessary research. It is expected that the major portion of the candidate's time will be spent in clinical or experimental research.

The candidate and the institution supporting his nomination are asked to supply the College with a planned program for the nominee. On completion of the research scholarship, the medical school or institution supporting the candidacy of the nominee is expected to absorb the research scholar into its faculty and support him on a full-time basis with its own budget.

A Committee on Selection has been appointed to pass on individual candidates, their program and the conditions of work as outlined by the sponsoring institution. Final arbiter in the selection of candidates will be the Regents of the American College of Surgeons.

Inquiries may be addressed to the Research Scholarship Committee, American College of Surgeons, 40 East Erie Street, Chicago 11, Illinois.